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An Address.[1]

By GILBERT BROWN, M.B., Ch.B. (Liverpool), D.A. (R.C.P. & S., England),
President, Australian Society of Anæsthetists.

IN opening this first session of the Australian Society of Anæsthetists I wish to thank the State representatives for the honour that they have conferred upon me by electing me to the position of President. I fully appreciate the responsibilities of the office and will carry out the duties to the best of my ability; but it will not be possible to fulfil the aims of the Society without the active and whole-hearted cooperation of all members. My first official duty is the pleasant one of welcoming the members and visitors who have come from all the States of Australia and from overseas. It is specially gratifying to find among our visitors Dr. Z. Mennell, senior anæsthetist of Saint Thomas's Hospital, who has travelled thirteen thousand miles to visit us and to represent the Association of Anæsthetists of Great Britain and Ireland.

[1] Delivered at a meeting of the Australian Society of Anæsthetists, September, 1935.

Formation of the Society.

It may be of interest to members to hear the story of the formation of this society. For a number of years the anæsthetists of Australia had felt that there was very little contact between them and that to work in isolation was good neither for the individual nor for anæsthesia. At the close of the session of the Section of Anæsthetics of the Australasian Medical Congress (British Medical

Association) which was held at Hobart in January, 1934, an informal meeting was held at Hadley's Hotel, Hobart. The matter was discussed by the representatives from all the States, and it was decided that an Australian society of anæsthetists should be formed. A skeleton constitution was drafted and Dr. Geoffrey Kaye was appointed temporary secretary. He was instructed to communicate with the anæsthetic sections of the British Medical Association in the States where they existed, asking them to appoint a representative; otherwise the Council of the Branch in that State was approached with a request that they nominate the most suitable person. In this manner an executive committee was formed, and it has gradually built up a constitution for the society. This has entailed much time and correspondence.

Membership.

It was decided that ordinary members must be members of the British Medical Association and that membership should be limited to those men who hold, or have held, posts as honorary anæsthetists in teaching hospitals, or those men who have rendered some special service to anæsthesia and who are approved by the executive committee.

Name and Form of the Society.

The name and form of the society gave rise to much thought, especially as to whether it should be a federal section of the British Medical Association or a separate body. It was finally decided that a separate society of anæsthetists would be preferable, although all ordinary members must belong to the parent Association. Some of the reasons which influenced this decision were:

1. Sections of the British Medical Association are open to all members of the Association, whether particularly interested in a specialty or not. The society aims to be an association of specialist anæsthetists.

2. Complete freedom of action is necessary for the society to obtain the best results. This might be hampered by having to be very closely associated with other British Medical Association activities. It might even limit the choice of time, place and procedure of meetings, or even the choice of guest speakers. The society is not responsible to anyone except itself, so far as its policy does not contravene the obligations which its members, as individuals, owe to the British Medical Association.

3. It might not be possible to affiliate with an overseas organization which had members who did not belong to the British Medical Association.

4. It was felt that the prestige of an Australian Society of Anæsthetists would be greater than that of a section of the British Medical Association when negotiating with public bodies or overseas organizations.

Objects of the Society.

The objects of the society are set down as follows: (i) to improve the status of anæsthesia in Australia; (ii) to facilitate the exchange of ideas between Australian anæsthetists and between them and overseas anæsthetic organizations; (iii) to encourage research into questions appertaining to anæsthesia; (iv) to arrange, if possible, with THE MEDICAL JOURNAL OF AUSTRALIA for the publication of at least one anæsthetic supplement annually, or one or more entire issues to be devoted to papers on anæsthesia.

Work has begun and some progress has already been made. New sections of anæsthesia have been formed in several States; memoranda have been circulated to members showing the results obtained with certain drugs and technical methods. A travel bureau has been instituted to advise members of the opportunities available for work when they are abroad. Further obvious activities are: to encourage and correlate clinical research; to approach the universities, hospitals and other bodies with reference to the teaching of students and the appointment and status of honorary anæsthetists; to endeavour to rectify the wide variation in type of coronial inquiry into deaths under anæsthesia; to combat the exceedingly high price of anæsthetic drugs.

The society will be able to give the considered opinion of the combined anæsthetists of Australia and so produce a weight of evidence that must command more respect than the voice of any one man working alone.

Anæsthesia in England.

Early this year I decided to visit England in order to gain further knowledge about many of these problems and to see something of the most recent anæsthetic work. During my absence the work of the society has been carried on most ably by Dr. F. W. Green, as Acting President, and by our Honorary Secretary, Dr. Geoffrey Kaye. In an address of this type it is obviously impossible to give an adequate review of the work seen overseas, and so I shall try to give you a short *résumé* of the points that were most interesting to me.

Gas Anæsthesia.

The most striking change in technique was the enormous increase in the use of the gaseous anæsthetics. One might say that England has become "gas-minded", as, with the exception of a few of the older men, some form of gaseous anæsthesia is considered the anæsthetic of choice for the majority of operations. This is not a passing phase, produced by young and experimental anæsthetists, as many of the surgeons demand this type of anæsthesia. The gynæcologists of the London Hospital now insist upon gas anæsthesia for their work, as they have found out by practical experience that their patients have a more comfortable convalescence and that the mortality is lower than when ether is used. A few surgeons informed me that it was somewhat more difficult to operate under gas and oxygen, and that they had found it necessary to make minor changes in their technique. They were, however, quite contented to put up with this difficulty, as they found that their patients had

less vomiting, fewer pulmonary complications and a more rapid convalescence. It seemed to me reminiscent of the introduction of rubber gloves into surgery. Those of you who are old enough to remember the introduction of rubber gloves, will also remember some of the older surgeons who boasted that they did not believe in this new-fangled idea, that it was not necessary, and that it made surgery more difficult and more dangerous. Yet today the use of rubber gloves is deemed a natural and obvious part of the operative technique.

The routine methods of workers in gas anæsthesia in England, I found, could be classified into three main types. The first type is pure nitrous oxide and oxygen anæsthesia, with little premedication and no addition of ether. Relaxation is produced by "secondary saturation" and, although this does not achieve the flabby abdominal wall, beloved of the gynæcologist, it is possible to employ it for the performance of any abdominal operation. This form of anæsthesia is used as a routine at the North Middlesex Hospital by de Caux, who has not even had an ether chamber attached to his machine for more than a year. Many anæsthetists, however, do not like secondary saturation, as the blood often becomes very dark in colour, and they fear that there may be danger of damage to the myocardium. Such men employ the second type, in which rather heavy premedication is employed; nitrous oxide and oxygen is then given and supplemented by a little ether or by an occasional very small amount of devinyl ether. Premedication is administered in much larger doses than those which are usually employed in Australia. "Omnopon", 0·045 gramme (two-thirds of a grain), and scopolamine, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), may be followed by a basal dose of "Avertin" before the gas anæsthetic is begun. This is sometimes supplemented still further by intravenous injections of "Evipan", "Nembutal", heroin or morphine. It is claimed that there are no deleterious complications following these enormous doses, as the percentage of oxygen given is so high.

The third type is that in which a large amount of ether is given, and the gases are used merely as a vehicle. The reasons for this particular method appear to be partly the use of inferior apparatus, partly the demand of some particular surgeon for an absolutely flaccid abdominal wall, and partly as a salve to the conscience of the anæsthetist whose patient has demanded gas.

Carbon Dioxide Absorption.

At present there is a fashion to fit gas machines with some form of apparatus to absorb the carbon dioxide in the exhaled gases. The nitrous oxide gas is unchanged and may be used again and again, so long as sufficient fresh oxygen is introduced into the mixture before it is again breathed by the patient. This method has the advantages of decreasing the cost of gases, lessening the weight of cylinders that have to be transported, and producing a quieter respiration. The absorbing agent is ether soda lime or a solution of caustic soda. There are many different forms of apparatus, all of which have their own advantages and disadvantages. The simplest type is that of Rowbotham, who merely places a handful of soda lime into a wide-mouthed rubber bag which is introduced into the circuit. Possibly the most complicated is the "Anæsthetor", invented by Primrose, of Glasgow, in which the gases pass backwards and forwards through a solution of caustic soda.

With all of the technical methods involving carbon dioxide absorption there seem to be three difficulties which are troublesome to overcome. It is not easy to produce an absolutely gas-tight joint between the patient and the machine. Unless the absorber is near the face-piece it is hard to avoid dead space, and consequently the carbon dioxide is not completely absorbed. Lastly, it is difficult to provide the exact amount of oxygen for basal requirements. If the amount of oxygen is too great, the depth of anæsthesia will be inadequate; if the amount of oxygen is too small, anoxæmia will occur. All this work is interesting and should eventually lead to a thoroughly practical and efficient machine, but at present there seems to be room for considerable improvement. Absorption methods have a special appeal to the Australian anæsthetist on account of the excessive price that he has to pay for nitrous oxide gas; this is more than three times that paid by his English colleague.

Cyclopropane.

Cyclopropane is much too expensive to use by ordinary flow methods, but when absorption technique is employed the amount of gas required is so little that the cost is relatively small. Cyclopropane has certain advantages over nitrous oxide gas, as it may be used with a percentage of oxygen as high as 80% to 85%; there is better muscular relaxation and the respiration is quieter and more shallow, a condition rather like that of chloroform anæsthesia. Nausea and vomiting are slightly greater than with nitrous oxide, but definitely less than with ether. The blood pressure is always raised during administration, usually about 20 millimetres, although at times it may be as much as 60 millimetres. It has the disadvantage of increasing bleeding, occasionally producing respiratory depression of considerable amount, and is followed more frequently by post-operative tachycardia and fall of blood pressure. This gas should be used only with carbon dioxide absorption methods and by an expert gas anæsthetist. Heavy premedication is not advisable with cyclopropane on account of respiratory depression. Some anæsthetists consider that its use is contraindicated in radical operations on the breast, in thyreoidectomy and in prolonged abdominal operations.

Devinyl Ether.

Devinyl ether is being used in England to a certain extent, chiefly as an adjuvant to gas anæsthesia. So far it has been difficult to obtain and is rather expensive. It appears to have distinct advantages over ethyl ether, as the amount required

is very small and full muscular relaxation is obtained as with chloroform. It is non-irritating to the lungs and does not appear to damage the heart or kidneys. It causes little disturbance of metabolism, but in view of the possibility of liver damage, similar to that associated with chloroform poisoning, administrations should not exceed one and a half hours' duration. The signs of anæsthesia are the same as with ethyl ether, but in long operations, especially in those on the abdomen, there are sometimes queer signs, such as jactitation. Rowbotham considers it is an excellent anæsthetic for Cæsarean section, when it is used with oxygen. Induction is very rapid, recovery, even from deep anæsthesia, is prompt and usually without any vomiting or other unpleasant after-effects.

Anæsthesia in Obstetrics.

During the last two years there has been a movement in England to provide anæsthesia for all women in labour. This has led to considerable research in an endeavour to find the best and safest method; and also to inquire whether it is safe for a midwife to give the anæsthetic without supervision. The anæsthesia should fulfil the following main requirements: (i) the procedure must be simple and inexpensive, (ii) there must be no danger to mother or child, (iii) there must be relief from pain during labour, (iv) uterine contractions must not be inhibited.

Chloroform capsules were introduced, but they have not proved very effectual, and one death at least has resulted from their use.

Paraldehyde by rectal injection has been used with considerable success at the Liverpool Maternity Hospital. A dose of seven drachms in seven ounces of olive oil is given, regardless of the weight of the patient.

Gas and Air.—The most interesting technique is Minnitt's method of inhalation of gas and air. The apparatus is an adaptation of the McKesson principle of intermittent flow of gases, and is controlled by the patient herself. When a pain commences she applies the face-piece and presses down a spring-loaded valve on it, which allows gas to flow. When anæsthesia is present the finger automatically relaxes, the gas is shut off and the mask falls away from the face. A fixed mixture containing 35% of nitrous oxide is employed. This method is very satisfactory and was seen in use in several instances. A large number of patients were questioned as to its effects in the relief of pain; both *primiparæ* and *multiparæ* were definite as to the amnesia that it afforded. The nursing staffs of different hospitals were enthusiastic about its use. If it was used in the very early stage, some slowing of the pains took place, but this did not occur after the os was dilated to the size of three fingers. Some patients were also given morphine, but the effects were better and more constant without it. The use of hyoscine was definitely contraindicated. In no case was there danger to either mother or child. The only disadvantages of the apparatus are that it is cumbersome, the cylinders are heavy, and the cost of nitrous oxide is considerable in a long labour. About thirty-five gallons of gas are used per hour; in Australia this represents an expense of four shillings and twopence per hour. Minnitt has recently introduced a portable model with light-weight cylinders; this has reduced the weight considerably, but unfortunately has increased the cost of the machine.

Spinal Anæsthesia.

Spinal anæsthesia varies in popularity in different centres, but some men use it extensively. The drug most commonly used is "Percaine", in the 1 in 1,500 hypobarbic solution popularized by the late Howard Jones. Nosworthy has introduced a modification of this technique, as he considers that sometimes there is an inefficient sacral block, although the analgesia is otherwise good. He injects 10·0 cubic centimetres of the 1 in 1,500 solution through the third lumbar space, the syringe is detached, and an injection of 0·8 cubic centimetre of a solution of 1 in 200 "Percaine" is made immediately with another syringe through the same needle. The patient is then turned on his face in the usual manner.

"Stovaine" is still used for low spinal analgesia if the duration of the operation is to be short. "Novocain" appeared to have few advocates, with the exception of Falkner Hill, of Manchester, who uses a solution of "Heavy Durocain". He considers that there is no danger from the absorption of "Novocain" into the blood in doses such as are usually injected into the dural sac. He has performed many experiments on animals and human beings to prove this. He was kind enough to lend me his M.D. thesis on the subject, in which he shows that the dose of "Novocain" required to bring about a fatal result, even by direct intravenous injection, is in the neighbourhood of 50 to 70 milligrammes per kilogram. The actual figures in one animal were 73·5 milligrammes per kilogram at the rate of 2·73 milligrammes per minute. In striking contrast to these enormous doses, he noted that 10·0 milligrammes or even 5·0 milligrammes injected into the fourth ventricle, or intracisternally, would kill a cat in about two minutes by paralysis of respiration.

One of the most interesting uses of spinal anæsthesia was that employed by Magill for lung lobectomy at the Brompton Hospital. He considers that it has made the former methods obsolete. He injects 14·0 cubic centimetres of 1 in 1,500 "Percaine" in the usual manner; the patient is then turned on his face with the upper dorsal spines as the highest point, both head and legs being kept at a lower level. This position is retained for five minutes, after which the patient is turned upon his side and the operation is begun. The patient is not turned upon his back at any time.

Toxic Goitre.

The majority of surgeons consider their operative results in toxic goitre have been definitely improved by the use of nitrous oxide and oxygen anæsthesia. Most anæsthetists use fairly heavy premedication, such as "Avertin" or large doses of "Omnopon" and

scopolamine, and follow this with nitrous oxide and oxygen; this is sometimes combined with the infiltration of the superficial tissues of the neck with local anæsthetic.

Joll, at the Royal Free Hospital, demands a rather peculiar technique, which is carried out by Morris. Premedication consists of morphine, 0·015 gramme (one-quarter of a grain), with atropine, 0·86 milligramme (one-seventy-fifth of a grain). Induction is with ethyl chloride and ether, administered by the open method on a mask covered with twenty-eight layers of gauze. The superficial tissues of the neck are then infiltrated with 50·0 cubic centimetres of a solution containing 0·5% of "Novotox" and 0·0005% of adrenaline. A rubber endotracheal tube is then passed, either through the nose or the mouth, and anæsthesia is continued with nitrous oxide and oxygen, without the addition of ether. Restraint straps are used upon both arms and legs. I was quite unable to find any reason for the use of ethyl chloride and ether during the induction period, except that it allowed more time and greater relaxation for the introduction of the endotracheal tube.

Dunhill, on the other hand, objects to an endotracheal tube, as he considers that it increases the amount of post-operative tracheitis, from which most of these patients suffer in some degree.

Langton Hewer, in a paper read to the Liverpool Society of Anæsthetists, related his experience of over one thousand anæsthetics for operations on toxic goitres. The technique that he has evolved from this large experience is to give "Omnopon", 0·022 gramme (one-third of a grain), with scopolamine, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), and to follow this one hour later with "Avertin" in a dose of 0·1 cubic centimetre per kilogram of body weight. Infiltration of the tissues of the neck is carried out with "Novocain", and a gas mask is fastened to the face with Clausen's harness. The mask is not, however, attached to the gas apparatus unless the anæsthesia is found to be too light. Hewer uses his own modification of Magill's apparatus with carbon dioxide absorption.

Fees and the Honorary System.

There is a growing feeling among English anæsthetists that the honorary system at hospitals is archaic and should be abandoned. At one time the number of anæsthetists was small and they were rewarded by receiving all the private work of the surgeon with whom they worked in the public hospital. This is no longer carried out so rigidly, and it has become more and more difficult for the anæsthetist to spend so much time doing unpaid work without even indirect recompense. Many anæsthetists and surgeons consider that there should be some direct reward for their work in public hospitals. This feeling has become intensified by the growing number of county council hospitals in which both surgeon and anæsthetist are paid for their work. The average fee seems to be about one guinea per hour. Already a few men have resigned an honorary appointment at a teaching hospital for a paid one at a county council hospital. This appears to be the thin edge of the wedge in the payment of doctors for hospital work. It is realized that this may mean the socialization of medicine and the problems which this entails, yet it seems to a large number of doctors that the time has come to abandon the honorary system. A system of salaried medical officers is being initiated for the first time in Australia at the Fremantle Hospital, which will be changed into a community hospital. The result of this experiment will be watched with interest.

ETHER IS NOT DEAD.

By Z. MENNELL, M.B. (London), D.A. (R.C.P. & S., England),
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WHEN your energetic Secretary wrote to ask me to address you, I was pleased and felt very honoured. I at once replied to accept and to find out what would interest you; he would not suggest a title for a paper, but when he was in England gave me a lead in a direction which had not previously appealed to me; perhaps it does not quite appeal to me now, as he told me I was getting old and my conservatism was well known. If thirty years of continuous teaching at a large teaching hospital means getting old, I am guilty, and I suppose the innate conservatism of increasing years and experience follows. I am conservative, and when I hear it stated at medical societies that ether is dead as an anæsthetic and it is reported in the medical Press that ether should not be used, it makes me see red.

Hence the title of this paper, "Ether is Not Dead"; and I hold myself briefed to defend ether and to make the case of interest to you all I am going to be provocative.

Thirty-four years ago, when I started to give anæsthetics seriously, the same cry was being raised about chloroform. Chloroform was dead and should not be used. I raised it myself and was in the fashion. Ether was the only anæsthetic which should be used, chloroform was a danger—all the same old tale. The local general practitioners could not give their own anæsthetics, as they had not been taught how to use ether—a boom for the specialists, in which I had my fair share. I suppose I use more chloroform now than I did then; believe me, it has its uses. Ether certainly has not replaced it in maternity work, and I know of no other anæsthetic so useful in those comparatively rare cases of high intracranial pressure when the respiration rate is very slow. Such cases I know are rare, but many come my way in my association with the National Hospital, Queen Square. So chloroform has lived thirty years since, according to the pundits, it was dead.

[1] Read at the annual meeting of the Australian Society of Anæsthetists, Melbourne, September, 1935.

Next we were told morphine and later scopolamine must always be used before any anæsthetic as a preliminary narcotic. I was rather more sceptical of this craze, but I realized at once the uses of the latter in certain classes of surgery. Scopolamine is almost forgotten now, but, used in the right cases, it is invaluable and is being used more freely.

The use of atropine and a clear airway were the next great advance in anæsthesia, about 1908, and these two necessities when using ether were at first looked at askance, then held as giving with ether the finest and safest anæsthetic ever found. The next stage was the abuse of this atropine-gas-ether sequence, as it was carried to great depths for very long periods—"deep ether" as Rood called it, and he claimed that with the use of quantities of oxygen it was safe to do anything for hours on end by this method. Of course, I adopted this method whole-heartedly, and the surgeons loved it; their job became too easy and they then did too much—more than they could have done with any other form of anæsthesia. I was lucky here, for I had very little trouble, as my surgeons were quick and never took more than an hour over the most difficult belly operation. But with increasing years, mine and theirs, their operations got slower and slower, one hour extending to three or four, and the method fell into disrepute on account of the number of chest conditions, the so-called ether-pneumonias, first the closed and then the open ether method being blamed. I am still convinced that deep ether gives the best form of anæsthesia for any belly operation lasting up to an hour. Those of you who have had experience of sewing up a really lax belly as against a stiff, quiet one, such as is obtained by scopolamine, will at once recognize what I mean. I have had that experience myself many times, and if all anæsthetists were made to sew up a quiet, stiff belly sometimes, it would make them more sympathetic to the surgeon and realize the trauma to which their patients were subjected. Thus up to 1912 ether reigned supreme, either following narcotics or not, according to choice, but with the almost universal use of atropine.

In 1912 the word barbiturate was first heard in conjunction with anæsthesia in England. Veronal had by that time passed through a craze of the universal hypnotic given by the mouth. Enormous doses were given and then a whisper began to go around that all was not well—an idiosyncrasy was talked about—and I personally saw some horrible cases. Then from Germany, via Russia, came hedonal, another barbiturate, and this was given intravenously as an anæsthetic. It was introduced by Page when he was resident assistant surgeon at Saint Thomas's. I took it up and a great many cases were done at that hospital. Again I shared in this boomlet and went all over the country demonstrating how to use hedonal intravenously and with a special apparatus. We were still at this work when the War came. Hedonal was suddenly unobtainable. I used isopral for a short time and, had this shortage not occurred, there is no doubt we should have been using smaller doses as a preliminary to a general anæsthetic, as I had already begun to do. But the same whisper was going round and becoming more outspoken; it was not the safe drug we were led to believe; the same word was heard: idiosyncrasy. I was fairly satisfied, and many of the complaints were due to faulty technique. But hedonal was unobtainable and we had other things to think of. I had had my lesson, however, and in doing so much intracranial work I was convinced of the necessity of the anæsthetist being out of the surgeon's way, both for his own comfort and the surgeon's temper. I then turned to my old friend ether again and tried it both by the rectum and intravenously; with neither method was I impressed, the main difficulty being that ether boils off any solution at about body temperature. So then I tried Kelly's machine for intratracheal administration of ether, which he had put on the market when he returned from America, it being a simplified form of what he had seen of Meltzer and Elsberg's machines. This machine really delivered air laden with ether vapour at the bifurcation of the trachea under a variable known pressure; I used it continuously, for heads especially, for nearly twenty years. I published papers about this method in *The Proceedings of the Royal Society of Medicine* and in America, most of the work being done with my old friend Sir Percy Sargent, who would have nothing else given for such cases. This was all very well, and was completely satisfactory until the slow-motion surgery was introduced from America, and it certainly was not satisfactory for operations lasting many hours, especially as diathermy was also part of this fashion. As you all know, the mixture of ether and oxygen is most terribly explosive. Ether and air are much less explosive, but still a real enough danger to be alarming. This difficulty was overcome by using a larger catheter and an expiratory valve at a distance from the face, thus eliminating pressure and the escape of ether vapour under the towels. Still, ether alone for many hours was not quite good enough and, instead of air, nitrous oxide and oxygen were adopted more as a vehicle than as an anæsthetic, and to enable the amount of ether used to be materially reduced. This is now my present practice, and I am satisfied until something else turns up, even though diathermy is no longer used at the National Hospital and the craze for slow motion is being dropped.

I had long before this discontinued the use of the heating chamber attached to Kelly's machine, as I could not convince myself it was of any value, and it was impossible to keep an even temperature of the water upon which it relied for its heat. I noticed I no longer got the marked sweating, and when at Saint Thomas's we did two series of cases, one with the vapour warmed (properly) and the other unwarmed, the incidence of bronchitis was higher in the former than in the latter. Many of the anæsthetists in France and in other fields during the War had but little experience, and when a machine was put on the market consisting of two junker bottles, the one a very large one for ether, and the other for chloroform, anæsthesia was found

to be easier and less irritation was obtained than when using plain ether. This machine had a warming chamber attached to it, and this was hailed as the important part of the machine; it was claimed that the percentage of chest sequelæ was materially lessened. It undoubtedly was, but not from warming the vapour, but from the proper use of chloroform. In the first place the length of rubber tube from all these machines must be very short, as the vapour loses its heat very rapidly; and if the tube is over two or three feet long, the temperature of the vapour, when it reaches the patient, is that of the theatre. Hence the old legal argument of justification became operative; the warming chamber did not do what was intended and, had it done so, it would have been harmful. Very few, if any, of the new machines make any provision for warming.

In cerebral surgery, morphine, scopolamine, local anæsthesia, intravenous use of alcohol and the newer barbiturates have been tried and, except in a few instances, have been dropped.

"Avertin", to my mind, stands in another category. I like it for head work, but the surgeons I work for will have none of it. Sargent was most emphatic.

Now what I have said about these various drugs has been in terms of what applies to brain surgery, the most interesting and difficult branch of the art, with special difficulties attached to each case; but what applies to this work, which intensifies the difficulties and dangers of anæsthesia, also applies in a minor degree to ordinary work.

The same old whisper is going around: all is not well with the barbiturates, their use is not devoid of danger, especially when used intravenously, and the same old word idiosyncrasy is cropping up. What is put into a vein is irrecoverable. Why should "Evipan", which is by far the quickest and easiest to give, be free from the same dangers as the others of the group? It most certainly is not, which is only to be expected, and I believe the whisper which is already heard will soon become a roar when some of the dangers are brought home more forcibly to the surgeons. The danger of the use of morphine, which intensifies their action, is now fairly well recognized.

I like "Avertin"; its use is easy and many patients like it. For all orthopædic work it is a boon. As a preliminary without morphine it is extremely useful before oral or nasal intratracheal insufflation with nitrous oxide and oxygen; better still with a little or our old friend ether. Just as much care in dosage must be used as with any other anæsthetic. Every patient must be seen beforehand and the correct "Avertin" weight must be estimated. To take two extremes: it would have been a fatal thing to give the acromegallic doctor of twenty-four stone, whom I had to tackle not long ago, twenty-four stone's worth of "Avertin"; or for the thin, extremely thyreotoxic woman of four and a half stone, four and a half stone's worth would have been equally futile. See the patient and estimate the "Avertin" weight and then give the appropriate dose. Underdo it rather than overdo it. The retrograde amnesia is very marked, and you always have ether to fall back upon. Contrary to general ideas, I have seen at least half a dozen people who have had several operations ask to be let off "Avertin" and to have an ordinary anæsthetic, by which they meant gas and ether; and you must all recognize that vomiting and headaches are not the rarities they are said to be after its use. Another use for "Avertin" is before the induction of local, or rather regional, anæsthesia. I believe this would have a more extended use if surgeons would only learn how to use a hypodermic needle. There are surprisingly few who can, and it is astonishing to me how bad many of them are at it. "Avertin" and spinal anæsthesia can be used satisfactorily, but only with great care and in a limited number of cases.

Now I must think of my subject, ether. Why is it under a cloud? I am confident that the cloud is only a passing one and that the sun, ether, will soon shine out again with renewed force and that there will perhaps be a better understanding of its limitations.

The main reasons for the temporary eclipse are, to my mind, three: (i) the slowness of modern surgical procedures; (ii) lack of skill and knowledge in its use; (iii) attempts to use the open method of giving ether after induction with chloroform or weak chloroform (two parts of chloroform and three parts of ether) or, again, alone.

Let us shortly look at these three points.

1. The thing which has changed anæsthesia is, of course, the requirements and demands of so many of the surgeons who have adopted deliberate methods of operating—the slow motion stunt—and which from an anæsthetist's point of view I dislike. As an ordinary doctor I cannot make myself believe there is any advantage in any way to the patients. It seems to me there is a danger of too much technique and no surgery.

The majority of you must agree that speed, other things being equal, is one of the greatest factors of safety in surgery. Your patients are much less ill after a half hour's operation than after one taking two hours, and recover much more quickly. To carry this to an extreme, it is much better to remove a lateral recess tumour in forty minutes to an hour (as I have seen dozens) than to perform decompression in nine hours, no matter what anæsthetic is used. It is not safe to give deep ether to a patient undergoing gastrectomy which is going to take three hours, but perfectly safe and, I believe, the best form of anæsthesia for one which takes an hour or under. So that in considering anæsthetics, old and new, we must consider the change which has come over surgery since the introduction of newer methods. I do not believe in these slows methods: two hours to open a skull, two hours to sew up a scalp, two hours for the removal of tonsils, two hours for a prostatectomy in a poor septic old man hung up in a Trendelenburg position. Naturally chloroform and ether have fallen into disrepute under these conditions, and hence the search for modern drugs and methods which will enable the surgeons to carry out their nefarious schemes.

The nitrous oxide-ether sequence is far the best anæsthetic for all operations lasting up to an hour, operations on the gall-bladder, gastrectomies and suchlike operations; but such is the nature of the anæsthesia that the surgeon trades upon it, does too much, and often takes double or treble the time necessary, and is lulled to sleep by the lax belly. I know the men I can use it for; they have no trouble, and the so-called pneumonias are very rare in their practice. There is no more speedy, pleasant and safe anæsthetic than ether and a Clover mask properly used with understanding for such men, and I am glad to think I have the support of an increasing number of anæsthetists who are doing an extensive practice.

2. The proper use of the Clover mask is no longer taught extensively. There is a knack in its use: the bag must not be used to limit the air supply, but as a reservoir of ether vapour. An empty bag means bad anæsthesia.

The use of chloroform and ether (two parts of the former and three of the latter) for induction is a quibble, and I am glad to know that the Council of the Association of Anæsthetists advised the Ministry of Health of their considered opinion that it was only dilute chloroform, and more dangerous than pure chloroform, as it often gave an increased sense of security which was fallacious.

If I have made out my case, you must understand that we cannot do without ether; in fact, if only one anæsthetic agent were allowed to be used, ether would be the one chosen. Practically everything which can be done by other means can, given sufficient skill, be done as well with gas and ether, and with more safety.

The objections to its use are well known: it is difficult to use, it has a disagreeable smell, and it is said to cause more severe after-effects than other anæsthetics. These objections, if well founded, are serious. Let us examine them from the point of view of my brief and I will give you, without any argument, the fact that ether without any other aid to producing anæsthesia is out of date. Now is it difficult to use? I most emphatically say "no". The ordinary student, after about twenty inductions, becomes quite passably efficient in the use of a Clover mask; some do so more quickly than others, and some will never acquire the knack, no matter how often they attempt its use. The ordinary routine that I teach is to give a big dose of atropine followed by nitrous oxide-ether sequence for any operation up to an hour, or longer if no great depth is required. In this way there should be no taste of ether during induction if the Clover machine is used and correctly fitted up. The machine should not be seen by the patient and there should be no stifling sensation. Unconsciousness is as rapid as with ordinary nitrous oxide, and ether should not be used until rebreathing has been started. I teach at the hospital that if it can be said of anyone that "he takes the anæsthetic badly", it can equally be said that "the anæsthetic is being badly given". There is this knack in using the machine, and with increasing experience fewer patients are met who show any resentment or discomfort with this method. A few breaths of carbon dioxide make full induction smoother and quicker. Again let me repeat: the bag must not be looked upon as limiting the air supply, but only as a reservoir of vaporized ether. A collapsed bag means a badly given anæsthetic and a complete failure to comprehend the proper use of a Clover inhaler. Push the ether as fast as is possible when regular breathing ensues; give ether until the pupils are dilated, and when the teeth are freed introduce a properly fitting artificial airway. When this is done, reduce the strength of the ether vapour until it is easily tolerated, and as soon as possible increase the strength until the shallow respiration with a bright pink colour indicates that the adductors of the vocal cords are paralysed. Then, and then only, relaxation of the muscles occurs and it is possible to maintain a sufficient depth of anæsthesia with very little ether. Keep a medium-sized pupil; if it gets smaller, give more; if it gets larger, give less. Recovery is quick, there is very little sickness, and the taste is not marked. When used for operations up to an hour, morphine, scopolamine, barbiturates, chloroform, all impair the efficiency of this sequence. Their use alters the whole type of anæsthesia. It seems to me that if this sequence were more frequently used, taught properly, there would not be the same cry for all these modern so-called basal narcotics.

Some of you may have noticed that I spoke very definitely of the paralysis of the adductors of the vocal cords. I can demonstrate this fact at any time, and am frequently doing so to the sceptical, and they are always convinced. David Ferrier showed that the abductors were paralysed before the adductors, leaving the latter unopposed. Those of you who use the intratracheal method of producing anæsthesia should note this fact, if you do not already realize it. When the patient is ready for the catheter to be passed, you can see straight down the trachea—the cords are poor little pink flapping folds of mucous membrane, and not the glistening white firm structures we are taught to look for. I dislike passing the modern large intratracheal catheter between these white cords, even during inspiration, as I think injury can and often does occur.

I have now arrived at another method of inducing anæsthesia: the intratracheal, which also gives a clear airway—a method again which appears to me to be much abused, abused in the way I have mentioned and by attempts to avoid the use of ether. Nitrous oxide and oxygen can produce a satisfactory anæsthesia for very long periods by this means after the use of a narcotic or basal narcotic; but a small dose of ether is usually given, and I believe firmly that it is advisable—ether again invaluable. I personally do not hesitate to give a few breaths of chloroform when necessary, especially in intracranial work; and always remember Horsley's urgent demand for more chloroform when he could not replace the brain after a decompression. I did not like it then, in the days before the intratracheal method was used, but Sargent, who did not ask for it and who hated chloroform like the plague, knew

this use of it, and would merely sniff and say that the pressure was falling in an unaccountable way.

Ether then, far from being dead, is very much alive. History is repeating itself. For anæsthesia up to an hour give as much as you like; over that time it is invaluable in much smaller quantities as a stimulant and aid to anæsthesia after preliminary narcotics. Those of you who have to look after patients following operations by various people must realize that speed is one of the greatest factors of safety in surgery; one might also add in ether anæsthesia.

This is my case for ether. I claim that ether is not dead.

BIOCHEMICAL ASPECTS OF ANÆSTHESIA.[1]

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THE biochemical problems of anæsthesia are numerous and the subject is intricate, but only the more important aspects will now be considered.

Our ability to visualize accurately the biochemical changes which accompany anæsthesia is due to the extraordinary advances in biochemical technique during the last three decades. Microchemical analysis has demonstrated many of the details of metabolic disturbance which accompany the administration of an anæsthetic, and it is to a consideration of these changes that I would ask your attention for a few minutes.

Blood Pressure Changes.

One of the chief anxieties of the anæsthetist is to maintain an efficient circulation during the period of anæsthesia. The welfare of the heart as the central pump and of the vasomotor centre as the chief controller of the tone of the arterioles is all important. A gross fall in blood pressure not only causes a poor coronary circulation, but also anoxæmia of the vasomotor and respiratory centres in the medulla, which is fraught with so much danger that it must be avoided at all costs.

The surgeon's knife is responsible for some of these biochemical disturbances, for the moment the first incision is made histamine, or a histamine-like body (H-substance) is liberated in the injured tissue. The greater the tissue damage, the greater the amount of histamine liberated. Histamine is very potent in inducing a fall in blood pressure, as it causes widespread dilatation of the capillaries and increased capillary permeability, so that fluid readily permeates the capillary wall and accumulates in the tissue spaces. In this way not only is there produced a diminished volume of circulating fluid, but the actual capacity of the circulatory system has been increased and hence the blood pressure falls. Fortunately the tissue damage induced by surgery not only liberates H-substance, but the nociceptive impulses initiated by the same procedure reflexly stimulate the suprarenal glands and that powerful vaso-constrictor adrenaline is liberated in increased amounts, and this tends to antagonize the action of histamine. Here we have a beautiful chemical mechanism which is in a condition of physiological equilibrium, provided the damage to the tissues is not too extensive. If, owing to pre-operative injury, the tissues are greatly damaged, then the liberation of H-substance may be so overwhelming that the adrenaline output may be insufficient to combat the dilator effect of the histamine and the blood pressure will fall and the symptoms of shock in varying degrees will ensue.

The anæsthetist should by the choice of the least toxic anæsthetic, for example, nitrous oxide and oxygen, and by care in its administration, cause the minimum amount of depression of the vasomotor centre and the minimum poisoning of the heart muscle. Ephedrin, owing to its comparatively prolonged action as a vaso-constrictor, may be useful in combating a fall of blood pressure.

Chemistry of Muscular Contraction.

A. V. Hill, Meyerhof, Lundsgaard and others have contributed greatly to our knowledge of the chemical changes occurring in muscle during its activity. These workers have shown that the contraction of muscle is normally an anaerobic phenomenon, but that the process of restoration is aerobic. During the anaerobic contractile phase glycogen in the muscle fibres is changed into hexose phosphoric acid (lactacidogen) and this is further converted into lactic acid and phosphoric acid. During the aerobic recovery phase one-fifth of the lactic acid is oxidized to carbon dioxide and water, and the energy so derived is used for the purpose of resynthesizing glycogen from the remaining four-fifths of the lactic acid which was formed. If the hydrogen ion concentration of muscle increases, the speed of oxidation and recovery decreases, and this is more marked in the case of cardiac than skeletal muscle. There is no doubt that chloroform anæsthesia causes a marked decrease in the glycogen content of heart muscle and an increase of its lactic acid content.

In one experiment the average glycogen content of the heart muscle of a control series of cats was 0·41%, while in another series, which had 2% chloroform administered for two hours, the glycogen had fallen to 0·1%. Thus a heart under the influence of chloroform not only has its glycogen store depleted unduly, but its contractile power is diminished by the accumulation of lactic acid which is not neutralized satisfactorily owing to the fact that the buffer content of cardiac muscle is less than that of skeletal muscle.

If the hydrogen ion concentration of the blood in the heart is increased, it causes dilatation of the coronary vessels, but this is even more marked in the case of anoxæmia. A fall in the oxygen saturation of the arterial blood may increase the coronary flow fourfold. Phosphagen is also a component of muscle

[1] Read at the annual meeting of the Australian Society of Anæsthetists, Melbourne, September, 1935.

and is broken down to creatine and phosphate during muscular contraction. The phosphate which is formed from phosphagen anaerobically unites temporarily with hexose to form hexose diphosphate. In the presence of oxygen the phosphate is liberated; it frees hexose and reunites with creatine to form phosphagen. In extreme fatigue of muscle lactic acid formation may continue, while phosphagen disintegration may stop almost completely. The glycogen content of nodal ventricular, of auricular, and of Purkinjé fibres of the heart increases in the order of fibres named, and it is interesting to record that the rate of conduction in those tissues increases also in the same sequence.

In toxic goitre, *diabetes mellitus* and in various cachectic conditions the glycogen content of the heart muscle may be greatly depleted, and hence it is of paramount importance that abundance of carbohydrate be administered pre-operatively to patients affected by such conditions.

Sudden death during the induction period of chloroform anæsthesia was demonstrated by Embley to be frequently due to vagal inhibition of the heart. The modern conception of vagal action is that it liberates at its terminals acetyl-choline, which causes cardiac inhibition. Loewi showed that stimulation of the vagus caused the liberation of a chemical agent in the frog's ventricle which could produce a vagal effect on another heart.

Acetyl-choline, when injected, reproduces the effect of stimulation of the parasympathetic system. Acetyl-choline is destroyed almost as soon as it is formed in the body by the action of an enzyme (esterase). This enzyme, which is found in all tissues and in the blood, converts acetyl-choline into relatively inactive choline. Weak concentrations of eserine will inhibit the action of the esterase.

Goodman Levy propounded a somewhat different theory as to the cause of sudden death in the early stage of chloroform anæsthesia. He demonstrated that injection of adrenaline into a cat anæsthetized with chloroform frequently caused ventricular fibrillation and death.

According to this view, sudden death of the human subject following the commencement of a surgical procedure while a patient is still under light chloroform anæsthesia is due to the surgical stimulus causing the liberation of adrenaline from the suprarenal glands and the subsequent development of ventricular fibrillation. It is becoming more generally recognized that the injection of adrenaline into a patient anæsthetized with chloroform is fraught with grave risks and has been the cause of many tragic deaths.

Control of Respiration.

The depth of respiration of the patient can be controlled by the anæsthetist owing to the fact that the respiratory centre is so sensitive to the carbon dioxide concentration of the blood. This is well exemplified in the well known table taken from the writings of the late E. H. Starling.

Percentage of Carbon Dioxide in Inspired Air.	Average Depth of Respiration.	Average Frequency of Respiration.	Ventilation of Alveoli with Inspired Air.	Percentage of Carbon Dioxide in the Alveolar Air.
0·04	673	14	100 (=6·6 litres per minute)	5·6
0·79	739	14	116	5·5
2·02	864	15	153	5·6
3·07	1,216	15	226	5·5
5·14	1,771	19	498	6·2
6·02	2,104	27	857	6·6

The use of carbon dioxide in the induction stage of anæsthesia is a practice that is now widespread and is sound in principle. The post-operative administration of "Carbogen" (5% carbon dioxide, 95% oxygen) is also based on accurate physiological knowledge. The control of external respiration is thus obvious, but that the anæsthetist influences internal respiration or tissue respiration is not so clearly recognized. The details of tissue respiration have not yet been clearly defined, but the following factors are probably of importance: (i) cytochrome, (ii) the respiratory enzyme of Warburg, (iii) dehydrogenases and glutathione.

Oxidation may be accomplished either by addition of oxygen or the removal of hydrogen. An auto-oxidizable substance is one which can unite with molecular oxygen at body temperature. Cytochrome, which is widely distributed in tissues, probably belongs to the hæmochromogen group of compounds. It exists in two forms, an oxidized and a reduced form. Reduced cytochrome is not auto-oxidizable, but under the influence of an enzyme, for example, an oxidase or the "respiratory enzyme" of Warburg, it undergoes rapid oxidation. It has been demonstrated that an oxidase-cytochrome system may rapidly oxidize the amino-acid cysteine to cystine, the cytochrome giving up its oxygen in the process and being converted to reduced cytochrome, which can again be oxidized under the influence of the oxidase. Neither oxidase nor cytochrome separately has this influence on cysteine. Thus:

(i) Reduced cytochrome + oxidase + O_2 → oxidized cytochrome.

(ii) Oxidized cytochrome + cysteine → reduced cytochrome + cystine.

The respiratory enzyme of Warburg is apparently an oxidase. It is an iron-containing compound, differing in composition from cytochrome, and seems an essential catalyst in the oxidation occurring in the tissues.

It must not be forgotten that removal of hydrogen may constitute an important form of tissue oxidation. Enzymes which are called dehydrogenases have been isolated from the tissues. They enable the hydrogen of the substance acted on (substrate) to be transferred to molecular oxygen to form water. Some of the dehydrogenases consist of complex systems of cooperating enzymes, whilst others seem simple in character.

Hopkins has isolated from the tissues a substance which he has named glutathione. It is thought that

it may facilitate oxidation in the tissues by acting as a hydrogen acceptor, the tissue substance being oxidized and glutathione reduced. Thus:

XH_2	+	G.S–S.G	=	2G.SH	+	X
Reduced tissue substance		Oxidized glutathione		Reduced glutathione		Oxidized tissue substance

The reduced glutathione is subsequently oxidized. Oxidation then in the tissues is under control of certain enzymes and their activity may be markedly depressed by deep anæsthesia. The very life of the tissues depends on these oxidative processes; hence it can be readily understood why prolonged deep anæsthesia is to be avoided.

Blood Sugar.

The normal blood sugar content of the fasting patient is approximately 0·1% (100 milligrammes per 100 cubic centimetres of blood). If 50 grammes of glucose be administered to such a fasting person, the blood sugar rises to 0·16% to 0·17% in the course of about 45 minutes and then declines, until in one and a half hours the fasting level is reached, or even a figure below the fasting level. Now it is well known that the administration of ether causes a rise in blood sugar, so that in diabetes the state of hyperglycæmia may be further aggravated by ether anæsthesia. Insulin, if administered in adequate doses some thirty minutes before the induction of anæsthesia, will maintain a comparatively normal blood sugar. If insulin is injected pre-operatively to a diabetic patient, it is desirable that blood sugar estimations be performed prior and subsequent to such an injection. If such estimations are not possible, then the insulin should be buffered by the administration of glucose in the proportion of approximately two grammes for each unit of insulin injected.

Acidosis.

Acidosis frequently follows the administration of an anæsthetic, particularly if there is much post-operative vomiting. Such acidosis is associated with a lowering of the alkali reserve of the blood. The chief cause of acidosis in such cases is a failure to metabolize the fats normally. The production of acetone, aceto-acetic acid and β-hydroxybutyric acid may occur. The body has elaborated most complex methods of combating such an acidosis, in which the blood, the liver, the kidneys and the lungs each play the most important rôles. The chemical factors involved in maintaining the normal reaction of the blood may be summarized as: (i) the bicarbonates of the blood, (ii) the phosphates of the blood, (iii) ammonia formation, (iv) the proteins of the plasma, (v) changes of base associated with hæmoglobin and oxyhæmoglobin equilibrium, (vi) transfer of chloride to corpuscles, (vii) bases derived from bone.

Bicarbonates of the Blood.

Of the bases in the blood plasma, in combination with weak acid, the major portion exists in the form of sodium bicarbonate; hence this constitutes the alkali reserve of the plasma. The reaction of the blood largely depends on the ratio $\frac{H_2CO_3}{NaHCO_3}$, which normally is as 1 to 19. Increase in H_2CO_3 or decrease of $NaHCO_3$ may give rise to acidæmia, diminution of H_2CO_3 or a rise in bicarbonate may induce alkalæmia. If both H_2CO_3 and $NaHCO_3$ increase or diminish proportionately, a normal reaction will be maintained. In acidosis the alkali of the blood is used to neutralize the acids present and the reserve becomes diminished. If a strong acid, such as hydrochloric acid, were introduced into the circulation, it would react with the sodium bicarbonate according to the following equation:

$$HCl + NaHCO_3 = NaCl + H_2O + CO_2$$

The sodium chloride formed in the reaction would be excreted by the kidneys. The water and carbon dioxide may be represented as forming H_2CO_3, which dissociates feebly, and hence has little influence on the hydrogen ion concentration of the blood.

$$H_2O + CO_2 = H_2CO_3 = H + HCO_3$$

Furthermore, the carbon dioxide (or slight increase of hydrogen ions) stimulates the respiratory centre and increases pulmonary ventilation, so that it automatically provides for its own removal by increased respiration. Some of this H_2CO_3 may interact with the K_2Hb in the blood corpuscles. The net result of the introduction of HCl into the circulation would be to deplete the blood plasma of some fixed base, but to cause practically no alteration in its hydrogen ion concentration. In a similar manner β-hydroxybutyric acid would be converted into sodium hydroxybutyrate and removed by the kidneys, while the carbon dioxide produced would be eliminated by the lungs. The hyperpnœa accompanying ketosis is thus a protective mechanism. Calculation has shown that an increase of two parts by weight of free carbon dioxide in a million parts of arterial blood is sufficient to double the breathing during rest.

Phosphates of the Blood.

Approximately four-fifths of the phosphate present in the body fluids is in the dibasic form (Na_2HPO_4). If hydrochloric acid be introduced into the circulation, it will combine in part with dibasic phosphate according to the following equation:

$$Na_2HPO_4 + HCl = NaCl + NaH_2PO_4$$

Thus the kidneys play a very important part in maintaining the neutrality of the blood, usually by excreting acid in the form of acid sodium phosphate.

Ammonia Formation.

Amino-acids, after absorption from the small intestine, are conveyed to the liver and in part deaminized, possibly by hydrolysis, or more probably by oxidation, with the liberation of ammonia.

Probably a little of the ammonia so formed combines with strong acid radicles and is excreted in the urine as ammonium salts, while the major

portion unites with carbon dioxide to form urea, which is excreted by the kidneys. Some biochemists consider that if a state of acidosis exists, the ammonia, instead of being converted into urea, is utilized for the neutralization of the abnormal fatty acids and that the ammonium salts formed are eliminated in the urine. According to the work of Benedict and Nash, ammonia, which they think is formed in the kidneys, acts in an indirect manner in combating acidosis and tends to conserve the fixed bases of the body, three-fifths of which are in combination with strong acid (hydrochloric acid) and are not available for purposes of neutralization.

Proteins of the Plasma.

Proteins consist of complex combinations of amino-acids which contain the basic radical NH_2 and the acid group COOH, and confer upon the protein molecule amphoteric properties.

The plasma proteins are chiefly combined with sodium, and this sodium can unite with any acid sufficiently strong to replace the protein molecule. Thus in the tissues where carbon dioxide is being produced in abundance, protein is displaced from its combination with sodium, according to the following equation:

$$\underset{\text{Sodium proteinate}}{NaPr} + H_2CO_3 = NaHCO_3 + \underset{\text{Protein}}{HPr}$$

The reverse process occurs as the blood passes through the lungs.

Changes of Base Associated with the Hæmoglobin and Oxyhæmoglobin Equilibrium.

Hæmoglobin exists in the blood in an oxygenated and a reduced form. Oxyhæmoglobin is approximately ten times as strong an acid as is reduced hæmoglobin. The hæmoglobin of the blood corpuscles is combined with potassium and the salts formed may be indicated as K_2Hb and KHHb.

In the presence of carbon dioxide in the tissues the following reaction occurs:

$$H_2CO_3 + K_2Hb = KHCO_3 + KHHb$$

The bicarbonate formed is actually an alkaline substance and the reduced hæmoglobin is only weakly acid and dissociates feebly. It has been estimated by Van Slyke that 85% to 90% of the base required to carry carbon dioxide in the blood is supplied directly or indirectly by the hæmoglobin. When carbon dioxide is diffusing into the blood from the tissues during metabolic activity, it displaces the weakly acidic hæmoglobin from its combination with potassium, according to the above equation.

When the blood reaches the lungs, the reduced hæmoglobin is oxygenated to the stronger acid HbO_2, and carbon dioxide is liberated in the blood corpuscles, diffuses into the plasma, and thence to the alveolar air.

$$KHCO_3 + KHHbO_2 = K_2HbO_2 + H_2CO_3$$

Thus hæmoglobin in its various combinations tends to prevent acidæmia in the blood flowing through the tissues, or alkalæmia in the blood in the lungs.

Transfer of Chloride to Blood Corpuscles (Hamburger Phenomenon).

By the transfer of chloride to blood corpuscles, details of which cannot be here discussed, some of the carbon dioxide is combined with the liberated sodium ions.

Bases Derived from Bone.

In extreme cases of acidosis, such as may occur in severe *diabetes mellitus*, the body may draw on the bases present in bone (calcium and magnesium) in an attempt to combat the acidæmia. With modern methods of treatment of diabetes this condition seldom occurs.

From the anæsthetist's standpoint it must be remembered that an adequate store of carbohydrate in the body is essential for the normal combustion of fat, and hence is of the greatest importance in the prevention of acidosis. Pre-operative starvation should be avoided and carbohydrate should be administered close up to the time of operation, so that glycogen in abundance may be present in the liver and muscles, particularly the cardiac muscle. If there is a special risk of acidosis, as in patients suffering from *diabetes mellitus*, then chloroform should on no account be administered, but nitrous oxide and oxygen or ethylene and oxygen should be chosen. Chloroform depresses liver function and tends to the development of acidosis. I should like again to emphasize that glucose enemata are probably of no value as a source of carbohydrate to the patient. Using dogs with sodium barbital hypnosis and ether anæsthesia, McNealy and Willems performed the following experiments: (*a*) they isolated the entire colon by ligatures placed round the ileo-colic junction and recto-sigmoid junction, (*b*) they isolated a loop of the distal part of the ileum. Into each of these segments of bowel glass cannulæ were inserted and the loops of bowel were carefully washed out with warm saline solution. In one series of experiments the sugar percentage was estimated in the blood obtained from the vein returning blood from the colon; 5% glucose was then introduced into the colon and samples of blood were again obtained twenty minutes and forty minutes later and the blood sugar was estimated. No rise in the blood sugar was detected. On the other hand, comparable experiments, in which 5% glucose was introduced into the loop of ileum, revealed a rise of 90 milligrammes *per centum* (0·09%), a figure similar to that obtained when glucose is administered orally.

Furthermore, Levi, experimenting on human subjects, found that glucose enemata containing 50 to 80 grammes of glucose administered in normal saline solution to normal fasting men caused a mean rise of blood sugar of 0·008%, whilst a similar quantity of glucose administered by mouth produced a mean rise of 0·085% of glucose, an increase more than ten times as great as when the sugar was administered rectally. It must be remembered that

a 5% glucose solution is practically iso-osmotic with blood. Therefore, if glucose is not absorbed from the colon, it will, by its osmotic action, greatly interfere with the absorption of water from this area of the alimentary tract. It is possible that in some patients regurgitation of portion of a glucose enema may occur through the ileo-caecal valve, and the sugar is then absorbed from the ileum, but it is probably true that in a vast majority of patients glucose is not absorbed when administered rectally. Glucose then should be administered orally or, if there is incessant vomiting, it may be given intravenously as a 5%, 10% or 20% solution.

I would conclude my remarks by referring to the rôle the biochemist plays in the determination of renal efficiency. Blood urea estimations, the urea clearance test and urea concentration test are individually and collectively of much value in estimating the efficiency of the kidneys, and hence the choice of anæsthetic, whether local or general. Particularly is this the case in patients undergoing prostatectomy. Sufficient has been said to indicate that a study of the biochemical changes in the patient prior to and during the administration of an anæsthetic has led to the evolution of a more rational anæsthesia.

SOURCES AND PHARMACOLOGY OF IMPURITIES IN ANÆSTHETICS.[1]

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IMPURITIES in anæsthetics and their probable effects have engaged the attention of chemists, anæsthetists and pharmacologists for so long a period, and technical improvements in their modes of preparation and purification have advanced to such an extent that a paper dealing with impurities is almost an anachronism.

Impurities are derived mainly from the following sources: (i) The crude chemicals involved in the process of manufacture; (ii) by-products, not completely removed by rectification; (iii) degeneration of the anæsthetic due to errors in storage and use.

This may be well illustrated by individual consideration of the main anæsthetics in current use.

Ether.

Ether is prepared by distilling under suitable conditions a mixture of ethyl alcohol or industrial methylated spirit and sulphuric acid, and rectifying the distillate.

In essence, the reaction consists of the subtraction of a molecule of water from two molecules of alcohol:

$$\begin{matrix} C_2H_5—OH \\ C_2H_5—O—H \end{matrix} —H_2O = \begin{matrix} C_2H_5 \\ C_2H_5 \end{matrix} > O.$$

Subsidiary reactions take place, with the production of other impurities, for example by the oxidizing action of the sulphuric acid a small quantity of the alcohol will be converted to acetaldehyde or, further, to acetic acid, while by complementary reduction of sulphuric acid, sulphur dioxide will be produced.

(*a*) $H_2SO_4 \rightarrow H_2O + SO_2 + (O)$

(*b*) $CH_3CH_2OH + (O) \rightarrow CH_3CHO + H_2O$
(Alcohol) (Acetaldehyde)

(*c*) $CH_3CHO + O \rightarrow CH_3COOH$
(Acetaldehyde) (Acetic acid)

Thus, before rectification, the ether will be contaminated with alcohol, water, aldehyde and acids.

Volatile impurities in the original alcohol, such as methyl and higher alcohols, and acetone, may be distilled with the ether, while the reaction of these extraneous alcohols with the sulphuric acid will produce small quantities of other ethers—dimethyl, methyl ethyl, methyl propyl, and others—together with corresponding aldehydes.

The bulk of these impurities will be of little interest to the anæsthetist because, with improved technique of rectification, they will appear only in the smallest traces in the product of reliable manufacturers. In this connexion it is worthy of note, in buying ether, that the British Pharmacopœia describes two grades—Æther and Æther Anæstheticus. The latter is a highly purified product, and any preparation labelled "Anæsthetic Ether" is required under the *Pure Food and Drugs Act* to conform to the pharmacopœial standard.

Of greater concern to the anæsthetist are the impurities developed in the ether by incorrect storage or by careless usage.

In contact with air or oxygen, especially with warmth and sunlight, such alcohol as is always present as a preservative is converted to aldehyde, while a percentage of the ether is converted to ether peroxide, which by hydrolysis will produce hydrogen peroxide.

The optimum conditions for decomposition are just those which are present in most forms of mechanical apparatus for delivering ether to the patient, air being passed over warmed ether.

The rate of formation of peroxides increases greatly as the degeneration proceeds,[1] so that residual ether from such an apparatus, added to a pure stock ether, is a potent factor for further degeneration. Such residual ether, though quite useful as a solvent or cleansing medium, should certainly be discarded for anæsthetic purposes.

Oxidation can be avoided by observing the pharmacopœial directions to store the ether in a dry bottle protected from light, in a cool place, and with well-fitting glass stopper or cork covered with tinfoil. As these requirements are not always within the realm of practical politics, protection from oxidation may be afforded by storing a small piece of copper in contact with the ether, or by adding a harmless oxygen absorber.

The copper, recommended by Nitardy and Bellheiner,[2] serves as a negative catalyst in preventing oxidation, but will not purify ether when once peroxides have been formed. Another preservative agent is hydroquinone, which is non-volatile and

[1] Read at the annual meeting of the Australian Society of Anæsthetists, Melbourne, September, 1935.

harmless, and is only required in small traces. H. O. Nolan[3] recommends as an efficient quantity two grains to the pound of ether (one in 3,500), while Middleton[4] regards a solution of one in 5,000 as sufficient.

It may be worth considering a few simple tests to indicate the presence of these impurities—tests which may be carried out without any special apparatus.

(*a*) *Evaporation.*—On evaporation of about two cubic centimetres on a clean watch glass no residue should be left, and no foreign or irritating odour should develop as the more volatile pure ether is dissipated.

(*b*) *Peroxides.*—Shake with a solution of potassium iodide in starch mucilage, in a small bottle filled to exclude air, when the development of a brown or reddish colour indicates traces; a blue colour shows grosser contamination.

(*c*) *Aldehydes and Acetone.*—Shake with an alkaline solution of potassium mercuric iodide in the conditions of the above test; no colour or turbidity should appear in five minutes.

Chloroform.

Chloroform is prepared by the action of chlorine, in the presence of an alkali on acetone, ethyl alcohol or industrial methylated spirit. Bleaching powder is often used as a source of both chlorine and alkali.

The primary reaction in each case is a chlorination of the acetone or alcohol, while the interaction between the chlorinated product and the alkali results in the formation of chloroform.

These reactions may be represented thus:

(*a*) From acetone:

$CH_3COCH_3 + 3Cl_2 = CCl_3COCH_3 + 3HCl$
(Acetone) (Trichloracetone)

$CCl_3COCH_3 + NaOH = CHCl_3 + CH_3COONa$
Trichloracetone + alkali → chloroform + sodium acetate.

(*b*) From ethyl alcohol:

$CH_3CH_2OH + (O) \rightarrow CH_3CHO + H_2O$
(Alcohol + oxidation → acetaldehyde)

$CH_3CHO + 3Cl_2 \rightarrow CCl_3CHO + 3HCl$
(Acetaldehyde) (Trichloracetaldehyde (chloral))

$CCl_3CHO + NaOH \rightarrow CHCl_3 + HCOONa$
(Chloral + alkali → chloroform + sodium formate).

Possible impurities from reagents and by-products may thus be alcohol, acetone, aldehydes, chlorine and hydrochloric acid, while from a subsidiary reaction between alcohol and hydrochloric acid ethyl chloride may also be present.

If the purified product be exposed to oxidation, as may happen by exposure to light and heat, or by agitation in partly filled or imperfectly stoppered bottles, a small quantity may be converted to phosgene or carbonyl chloride.

$CHCl_3 + (O) \rightarrow COCl_2 + HCl$
(Chloroform) → (phosgene)

As alcohol is more subject to oxidation than is chloroform, and as the aldehyde so produced is much less harmful than phosgene, quantities of dehydrated alcohol from 1% to 2% are always added as a preservative. This not only protects the chloroform, but if any phosgene be present, the alcohol destroys it with the formation of ethyl carbonate.

$2C_2H_5OH + COCl_2 = (C_2H_5)_2CO_3 + 2HCl$
(Alcohol + phosgene = ethyl carbonate).

Ethyl Chloride.

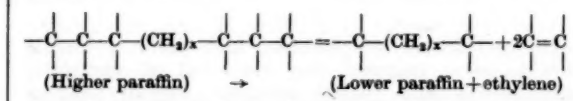
Ethyl chloride is prepared by the distillation of ethyl alcohol or industrial methylated spirit with hydrogen chloride.

The main impurities from manufacture—alcohol, hydrogen chloride and water—are readily removable in the actual distillation plant, owing to the great volatility of the ethyl chloride, which is gaseous at ordinary temperature and pressure. If ethyl chloride be made from industrial methylated spirit, a small and variable quantity of methyl chloride will be present.

Ethylene.

Ethylene, an unsaturated and inflammable hydrocarbon, the lowest member of the olefine series, is obtained from the products of decomposition of petroleum.

The heavy fractions of the petroleum oil are "cracked" by exposure under pressure, in a non-oxidizing atmosphere, to a temperature of about 460° C. The reaction may be represented graphically thus:



In this process, traces of higher olefines, together with acetylenes, are produced at the same time.

From the presence of small quantites of other bodies in the petroleum, including oxidized hydrocarbons and other compounds of oxygen, sulphur and phosphorus, such impurities as carbon dioxide, carbon monoxide, hydrogen sulphide, sulphur dioxide and phosphine appear with the ethylene and are removed with suitable chemical absorbents.

Official tests require that anæsthetic acetylene shall be at least 98% pure, as shown by the absorption of 98% of the gas when a measured quantity is exposed to fuming sulphuric acid or bromine.

Any residual gas can be tested for carbon monoxide and other impurities.

A simple test for carbon monoxide which is by far the most dangerous impurity, depends on its capacity to form a stable compound with hæmoglobin. The gas is shaken with a small quantity of diluted blood, which is then treated with a mixture of equal parts of pyrogallol and tannic acid, and allowed to stand. Normal blood forms a red precipitate which becomes greyish-brown, but with carboxy hæmoglobin, the precipitate remains red, the intensity of the colour being proportional to the amount of carbon monoxide present.

Nitrous Oxide.

Nitrous oxide (*nitrogenii monoxidum*) is prepared by heating ammonium nitrate:

$$NH_4NO_3 \rightarrow N_2O + 2H_2O$$

(Ammonium nitrate) (Nitrous oxide)

During the heating, a degree of dissociation together with auto-oxidation and reduction gives rise to such contaminants as nitrogen, higher oxides of nitrogen and ammonia or free acids of nitrogen.

Traces of dust and organic matter in the nitrate will give rise to carbon monoxide and carbon dioxide. The Pharmacopœia allows a maximum of carbon monoxide of 50 parts by volume per million of gas—a quantity which is disproportionately high.

Of other impurities, water is unobjectionable on physiological grounds, but when present in an undue proportion may freeze and block the valves as the released gas expands, while atmospheric gases are usually present up to about 6%. Official tests exclude the hydrides of arsenic, phosphorus and sulphur which may arise from impure ammonium nitrate.

Nitrous oxide is readily recognized by its characteristic odour and by its capacity to ignite a glowing splinter. As it shares this latter character with oxygen; it is distinguished from oxygen by its inability to produce the coloured fumes of nitrogen peroxide, when exposed to nitric oxide.

With this formidable list of actual or potential impurities in each anæsthetic, it is little wonder that such foreign substances have been viewed with grave suspicion when any uncalculated effects have been produced in practical anæsthesia. Nevertheless, very little actual experimental evidence has accumulated to substantiate these suspicions.

The unreliability of clinical impression in this respect has been demonstrated repeatedly by exhaustive chemical investigation of individual samples of anæsthetics which have been found unsatisfactory in use. The Medical Research Council has at different times undertaken to examine such samples, with entirely negative results with regard to the presence of poisonous substances or gross impurities.[5]

In assessing the pharmacological effects of impurities, the following factors contribute considerably to the difficulty: (i) The small quantity of impurity present, and its relatively small influence in comparison with the gross effects of the pure anæsthetic; (ii) individual variations in the response of patients to the same anæsthetic; (iii) apparent variations in the toxicity of the same anæsthetic on organs (heart, lungs, liver, kidneys *et cetera*) and on the acid-alkali balance; (iv) underlying condition of the patient with regard to toxæmia, anæmia *et cetera*; (v) mode of administration of anæsthetic; (vi) premedication.

It is interesting to recall the controversy which raged some years back round the subject of pure *versus* impure ether, when some observers, particularly Cotton in the United States of America, and Mackenzie Wallis in England, maintained that pure ether was not an efficient anæsthetic, its virtues being due to impurities, including dissolved gases—carbon dioxide, ethylene and ketones.

King and others[6] tried the effects of pure and of the recommended impure ether on cats, and noted less irritability of the trachea with pure ether, while less was required to maintain the anæsthesia. This work was confirmed by Stehle and Bourne.[7]

From their pharmacological effects, impurities may be regarded as falling into the following groups, which somewhat overlap:

(1) Anæsthetics: Alcohols (ethyl and higher alcohols); ethers (dimethyl, methyl ethyl, methyl propyl, and others); ketones (acetone, methyl ethyl ketone *et cetera*); hydrocarbons (acetylene and propylene in ethylene).

(2) Irritants: Hydrogen chloride, phosgene, aldehydes, peroxides, nitric oxide, chlorine, sulphur dioxide.

(3) Toxic: Carbon monoxide, hydrogen sulphide, ethyl sulphide, phosphine, sulphur dioxide, formaldehyde.

The members of the anæsthetic group have not been shown to produce any harmful effects, and, indeed, individually most of them have been used experimentally or practically as anæsthetics, without the production of undue irritation or toxæmia.

Though the ketones have often come under suspicion, it is known that workers with acetone, who necessarily inhale measurable quantities, do not appear to develop any toxic symptoms, while Wallis and Hewer,[8] in that fruitless controversy of impure *versus* pure ether, claimed that acetone with other impurities was "synergistically" beneficial. Wesley Bourne,[9] in a series of careful experiments on dogs, finds that ketones (di-ethyl ketone, ethyl methyl ketone and acetone) in anæsthetic ether produce no effects in concentrations up to 5%. The main significance of ketones as impurities lies in the fact that they are not normal degradation products, and so may serve as an indication of grosser contamination.

As has been previously indicated, alcohol is occasionally added, especially to chloroform and ether, as a stabilizer; and until it is completely destroyed it bears the brunt of any oxidizing actions, thus protecting the anæsthetic.

The irritants must be considered more seriously, because of their local, reflex and general effects.

The local effects, as shown by Henderson and Haggard,[10] depend on their solubility in water. If soluble, as in the case of aldehydes, peroxides, hydrogen chloride, ammonia and chlorine, they dissolve in the moisture of the walls of the upper air passages and produce local irritation and inflammatory reaction. Mendenhall and Connolly[11] note that pure ether will stop temporarily the ciliary activity in oysters, while the addition of aldehydes and peroxides produces permanent inactivity. They infer from this a comparable action with the ciliary epithelium of the larynx with a corresponding interference with its normal capacity to dislodge mucus and bacteria.

From the local irritation, a reflex vagal stimulation may cause a constriction of the bronchi and excessive bronchial secretion. These are undoubtedly factors in the production of post-anæsthetic pneumonia. Another vagal effect that may be produced is a temporary slowing of heart and respiration followed by rapid and irregular respiration.

Most of these reflex effects are, however, produced by the irritation of the anæsthetic apart from impurities, and are anticipated and minimized by the pre-anæsthetic administration of atropine. Bourne,[9] in his experiments on dogs, finds that ether contaminated with 1% of acetaldehyde produces respiratory embarrassment with variable blood pressure, tending in general towards depression, while with concentration up to 0·5% very little change was observed.

With peroxides, 0·5% produced respiratory disturbance (slow and forceful) with lowered blood pressure. With 0·3% no noticeable effects were recorded.

Non-soluble irritants such as phosgene or carbonyl chloride have little effect on the upper air passages, but collect in the bronchioles and alveoli, where, by decomposition, they may produce local inflammatory or toxic effects.

Thus, phosgene decomposes with the liberation of hydrochloric acid and tends to produce pulmonary œdema, with patchy bronchiolar necrosis if in sufficient quantity.

$$COCl_2 + H_2O = 2HCl + CO_2.$$

The latent period between its inhalation and subsequent hydrolysis increases its danger.

Nitric oxide as a contaminant of nitrous oxide behaves similarly, being oxidized to the peroxide, which in solution forms nitrous and nitric acids.

$$2NO + O_2 = 2NO_2$$
(Nitric oxide) (Nitrogen peroxide)

$$2NO_2 + H_2O = HNO_2 + HNO_3$$
(Nitrous) (Nitric acid)

While reflecting on the effects of the inhalation of gaseous irritants, and many anæsthetics apart from their impurities come into this class, the hyperventilation of the lungs, which so often occurs in the early stages of ether anæsthesia, may be a factor in the production of ether convulsions.

The effect of the forced respiration, with its resultant loss of carbon dioxide, tends to disturb the acid base balance of the blood, and to produce a degree of alkalosis. If the pathological condition underlying the surgical interference be associated with a similar disturbance, the summation of effects may conceivably produce a severe alkalosis, in which, as is well known, tetany and convulsions may occur.

Various impurities have been blamed for the production of ether convulsions, but without sufficient justification. S. R. Wilson[12] has suggested aldehydes and peroxides as causative factors, and is supported by A. C. R. Walton.[13] Bourne[14] regards these as being insignificant, while Hadfield[15] reports their appearance with chemically pure ether, and, in excluding excess carbon dioxide, anoxæmia and atropine as factors, notes their constant association with heat, being prevalent in warm weather and with warmed ether.

These, of course, are just the conditions which favour decomposition, but an examination of the residual ether after an anæsthetic associated with convulsions has failed to detect impurities.

L. A. Riddell, in a letter to *The British Medical Journal*,[16] makes the following significant observation:

> Lately, I had begun to use oxygen with a Shipway, and on two occasions I saw facial twitchings. The coincidence of the appearance of twitchings with my first use of oxygen instead of air struck me forcibly, so on the second occasion I used carbon dioxide as well.
>
> Again it may have been only coincidence, but with pure oxygen, twitchings appeared; on changing to carbon dioxide they went, but returned once more with the oxygen. It really seemed as if they were heralded each time by the appearance of a bright pink colour.

Other correspondents had reported the appearance of convulsions with coincident use of oxygen.

These observations seem compatible with the view that alkalosis, associated with carbon dioxide reduction, may be a factor. This is, however, merely an armchair reflection, as I know of no pharmacological observations to support it.

Of the toxic impurities, carbon monoxide stands out as one of the most dangerous and insidious. Were it not for its capacity to form a stable compound with hæmoglobin, it would be an indifferent non-poisonous gas, but its affinity for hæmoglobin has been estimated at from 300 to 400 times as great as that of oxygen.

A concentration of one in 10,000 is sufficient to convert half the hæmoglobin in the blood into a compound which is useless as a carrier of oxygen.[17]

Its danger lies in the fact that the increasing anoxæmia does not cause any respiratory distress, and develops so quietly that the anæsthetized subject glides almost imperceptibly into the danger zone without marked symptoms except the characteristic cherry red colour of lips and skin.

The carbon monoxide is not burned in the body, and is only slowly replaceable by oxygen. Anoxæmia, therefore, persists for a considerable time even with administration of oxygen, which is more effective if given under pressure.

Recovery is associated with some of the following symptoms—severe headaches, amnesia and neuritis, associated with degenerative processes in the central nervous system, albumin and bile pigments in urine, bronchopneumonia, sweating *et cetera*.[18]

Sherman and others[19] record a fatal case from carbon monoxide in ethylene, in which, after twenty minutes' anæsthesia, the blood showed a 60% saturation.

Other toxic impurities, such as hydrogen sulphide, phosphine, ethyl sulphide and sulphur dioxide, have such distinctive and arresting odours that they announce themselves and so condemn their vehicle as an anæsthetic.

Conclusion.

In conclusion, I should like to stress the following points:

1. Technical improvements in manufacture have reduced probable impurities almost to vanishing point.

2. With reasonable care in handling and storing anæsthetics impurities from degeneration will be considerably minimized.

3. With such care, impurities which may develop have not been shown to be grossly harmful, but in general the purer the anæsthetic, the smoother will be the induction and the less will be required for maintenance, with a minimum of after effects.

References.

(1) F. H. Carr: "'Ether Clonus': Aldehyde and Peroxide", *The British Medical Journal*, October 8, 1927, page 664.
(2) F. W. Nitardy and E. C. Bellheimer: "Mechanism of Stabilization of Ether by Copper", *The Journal of the American Pharmaceutical Association*, Volume XXI, February, 1932, page 112.
(3) H. O. Nolan: "Decomposition of Anæsthetic Ether: Simple Preventive", *The Lancet*, July 15, 1933, page 129.
(4) G. Middleton: *The Pharmaceutical Journal and Pharmacist*, 1924.
(5) Report of the Medical Research Council, 1923-1924; *ibidem*, 1926-1927.
(6) H. H. Dale, C. F. Hadfield and H. King: "The Anæsthetic Action of Pure Ether", *The Lancet*, Volume I, 1923, page 424.
(7) R. L. Stehle and W. Bourne: "The Anesthetic Properties of Pure Ether", *The Journal of the American Medical Association*, Volume LXXIX, July 29, 1922, page 375.
(8) H. J. M. Wallis and C. L. Hewer: *The British Medical Journal*, April 16, 1921, page 565.
(9) Wesley Bourne: "On the Effects of Acetaldehyde, Ether Peroxide, Ethyl Mercaptan, Ethyl Sulphide, and Several Ketones—Dimethyl, Ethyl Methyl, and Diethyl—When Added to Anæsthetic Ether", *The Journal of Pharmacology and Experimental Therapeutics*, Volume XXVIII, 1926, page 409.
(10) Henderson and Haggard: "Noxious Gases", American Chemical Society Monograph, 1926, Number 35.
(11) Mendenhall and Connolly: *The Journal of Pharmacology and Experimental Therapeutics*, Volume XXV, 1925, page 145.
(12) S. R. Wilson: "'Ether' Convulsions", *The Lancet*, May 28, 1927, page 1117.
(13) A. C. R. Walton: "A Note on Ether Convulsions", *The British Medical Journal*, July 7, 1928, page 8.
(14) Bourne: *The Journal of the American Medical Association*, January 29, 1927.
(15) C. F. Hadfield: "Discussion on Late Ether Convulsions", *Proceedings of the Royal Society of Medicine*, August, 1928, page 33.
(16) L. A. Riddell: *The British Medical Journal*, November 3, 1934.
(17) A. J. Clark: "Applied Pharmacology", 1933, page 386.
(18) Erich Leschke: "Clinical Toxicology".
(19) W. O'N. Sherman, C. M. Swindler and W. S. McElroy: "Carbon Monoxide Poisoning Following Ethylene Anesthesia", *The Journal of the American Medical Association*, June, 1926.

USES OF CARBON DIOXIDE IN ANÆSTHESIA.[1]

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MY purpose is to stress the usefulness of carbon dioxide in all varieties of anæsthesia and post-operative conditions and its importance in physiological processes. Forty-five years ago the Swiss physiologist, J. N. Miescher, in a review of all previous investigations, made the prophetic remark: "Over the oxygen supply of the body carbon dioxide spreads its protecting wings."

We have a very early mention of carbon dioxide and oxygen being used in resuscitation in II Kings, iv, 34, when Elisha brought back to life the son of the Shunammite woman. In the middle ages midwives used to apply their mouths to a baby's mouth and nostrils for a similar purpose.

When Clover's ether inhaler came into use it was recognized that the carbon dioxide in the expired air stimulated breathing. The *"Aetherrausch"* of the Germans, using a large wire frame mask made air-tight, gave a light anæsthesia after about twelve breaths; the same thing happens in the closed ethyl chloride method. In these cases there is also lack of oxygen.

About 1906 there was a great change over to the open administration of ether with little or no carbon dioxide conservation. There were soon attempts to make the administration semi-open or perhalational, giving more or less rebreathing of carbon dioxide.

About twenty-five years ago, Haldane, Priestley and Douglas began their epoch-making work on the part carbon dioxide plays in respiration. Yandell Henderson, Haggard, Corryllos and Birnbaum took up the matter in America, applied the use of carbon dioxide to carbon monoxide poisoning and functional depression after operations and other conditions. By 1921 carbon dioxide was being used in anæsthesia, though on the whole clinicians were slow to accept and apply the discoveries of the physiologists. Ogston,[1] of Aberdeen, describing his perhalation inhaler with raised sides in 1922, said:

> No doubt there was some concentration of carbon dioxide, but rarely of such a degree as to do any harm. Occasionally I saw shallow breathing and duskiness which might be attributable to this cause.

Quite early pure oxygen was used when there was considered to be insufficient oxygen intake, but the idea of leading in carbon dioxide from an outside source seemed strange or unnecessary, and only came later. One comes across such references in the literature as one in 1921 in which Ley,[2] a German, speaking of the administration of ethyl chloride, stated that he used a band tied tightly around the thighs so as to diminish the circulation; he held that if danger of intoxication subsequently developed, the bandage could be loosened, and the blood, which was rich in carbon dioxide, served for stimulating the respiratory centre. In 1925 Yandell Henderson[3] lectured in England before the Royal Society of Medicine on the use of carbon dioxide in anæsthesia. By 1925 several articles had appeared, mostly in American journals, referring to the benefits of carbon dioxide in anæsthesia and in post-operative complications. In 1928 E. Fischer[4] in Germany reported its satisfactory use in 300 abdominal and vaginal operations.

To expound the effects of carbon dioxide in the body is more the province of a professor of physiology, so, if you will permit, I shall quote freely from a recent review by Professor Samson Wright.

Professor Wright[5] considers that there is no need to adopt Lumsden's artificial subdivision of the respiratory centre. Normally the centre acts as a coordinated unit; if parts are cut off, the portion left can no longer control breathing normally. When an animal is dying from asphyxia, overdose of anæsthetic or interference with the medullary blood supply, it may be noted that, as the breathing fails, it sometimes passes through the stages of slow, prolonged inspirations and then of gasps; if the animal is revived, breathing recovers in the opposite order. Wright considers that the respiratory centre possesses an inherent rhythmicity, analogous to that of the sino-auricular node.

If air containing excess of carbon dioxide is breathed the pulmonary ventilation increases in a very striking manner. As the carbon dioxide tension in the alveolar air rises, it also rises in

[1] Read at the annual meeting of the Australian Society of Anæsthetists, Melbourne, September, 1935.

the arterial blood leaving the lungs. As breathing is stimulated, alveolar carbon dioxide is washed out, but as more of it comes in with each inspiration its tension in the alveoli can never fall quite to normal. So the arterial blood continues to contain a little more carbon dioxide than it does at rest. These facts are well shown in Table I from Haldane and Priestley. It will be noticed that the breathing increases first in depth and later in frequency, the increase in depth contributing considerably more to the increased ventilation. The limits of the compensatory mechanism are also shown. If the carbon dioxide in the inspired air exceeds that in the alveoli, for example, 6·02%, no possible degree of ventilation can keep the alveolar carbon dioxide below that figure. As carbon dioxide is also arriving in the venous blood, the alveolar carbon dioxide may rise higher still, for example, to 6·6%.

TABLE I.

Percentage CO_2 in Inspired Air.	Average Depth of Respiration.	Average Frequency per Minute.	Ventilation of Alveoli with Inspired Air.	Percentage CO_2 in Alveolar Air.
0·04	673 c.cm.	14	100 (taken as normal.)	5·6
0·79	739 c.cm.	14	116	5·5
2·02	864 c.cm.	15	153	5·6
3·07	1,216 c.cm.	15	226	5·5
5·14	1,771 c.cm.	19	498	6·2
6·02	2,104 c.cm.	27	857	6·6

Mode of Action of Carbon Dioxide.

Recent research has shown the mode of action of carbon dioxide to be complex. Carbon dioxide acts mainly on the respiratory centre, but it also acts partly reflexly by stimulating sensory nerve endings in the carotid sinus and the aortic arch, thus sending excitatory impulses to the centre. Heymans demonstrated this in 1927. The response to carbon dioxide is usually reduced by denervation of the carotid sinus (see Figure I).

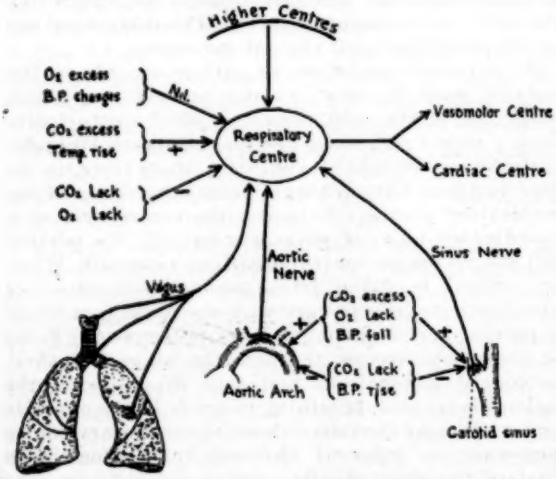

FIGURE I. Regulation of respiration (Samson Wright).

Changes of hydrogen ion concentration of the blood due to other causes than increased carbon dioxide formation in the tissues or excess of carbon dioxide in the inspired air readily affect respiration in order to preserve the appropriate blood reaction. In acidæmia hyperventilation washes carbon dioxide out of the blood and the hydrogen ion concentration is reduced to its proper level. Conversely, in alkalæmia breathing is depressed, carbon dioxide is retained in the blood as H_2CO_3 to bring the pH value to its correct level.

Oxygen Lack.

Severe oxygen lack very suddenly produced, as by breathing an inert gas, cannot be coped with; unconsciousness and death follow quickly. Less severe lack down to 14% stimulates breathing, but to nothing like the same extent as a small excess of carbon dioxide. The hyperpnœa washes out carbon dioxide, producing alkalæmia, and raises the oxygen tension till apnœa comes on with a tendency to periodic breathing. If the oxygen lack is of very gradual onset, the body responds to the alkalæmia produced, by the kidney secreting a less acid urine and less ammonium, thus keeping the blood reaction at the right level. The stimulation of respiration which may be produced by moderately severe anoxæmia is wholly reflex, and depends entirely on the integrity of the carotid sinus nerves and vagi (see Figure I). With the buffer nerves eliminated, oxygen lack acts as a depressant to the "isolated" respiratory centre. Also anoxæmia depresses the response of the "isolated" respiratory centre to carbon dioxide. Lack of oxygen does not sensitize the respiratory centre to carbon dioxide, as the isolated centre, with sinus nerves and vagi cut, responds more vigorously to carbon dioxide alone than to carbon dioxide excess combined with oxygen lack.

In a recent paper, entitled "Is Breathing Fundamentally a Reflex Phenomenon?" Gesell and Moyer[6] state that carbon dioxide under otherwise normal conditions may produce its relatively slow breathing by depressing the vagal accelerating function by chemical vagotomy, and that anoxæmia may produce its relatively rapid breathing by augmenting this function.

Blood Pressure Changes.

After section of the aortic and sinus nerves, blood pressure alteration no longer affects breathing. This shows that blood pressure changes (so long as they do not lead to severe cerebral anæmia) have no direct effect upon the respiratory centre.

The Vagi.

The vagal afferents which come from the laryngeal mucous membrane are concerned with the cough reflex. The afferent fibres from the substance of the lung undoubtedly regulate the rhythm of breathing; cutting the vagi abolishes the normal rhythm. The vagi signal the degree of expansion of the lungs; normally, when inspiration has proceeded to a certain degree, afferent impulses are set up in the vagi, which check inspiration, and consequently expiration takes place. This is generally known as the Hering-Breuer reflex. Afferent impulses are set up only very exceptionally during expiration.

Nerve Control.

The endings of nerves in the carotid sinus and aortic arch are sensitive to changes in blood pressure, carbon dioxide tension, hydrogen ion concentration, and the oxygen content of the blood. A fall of blood pressure and a rise in carbon dioxide tension or in hydrogen ion concentration and oxygen lack stimulate the respiratory centre. Conversely, a rise of blood pressure and a fall of carbon dioxide tension or hydrogen ion concentration reflexly depress it. The nerve endings of the carotid sinus lie in thin-walled vascular channels in the carotid body.

McMechan[7] maintains that adrenaline is necessary for the occurrence of normal physiological respiration; there is often lack of it in acutely ill patients.

The gaseous exchange in respiration is under such delicate control that, according to Haldane and Priestley:[8]

> Not only is oxygen supplied and carbon dioxide removed, but these processes are coordinated at every stage among themselves and with other physiological activities in a manner which is characteristic for each organism.

Some Respiratory Phenomena.

Apnœa is not due to excess of oxygen, as can be demonstrated by observation on hibernating animals, the respiratory movements of which become more frequent if they are warmed. The excitability of the animals is much depressed. Breathing appears to start again as soon as the respiratory centre is stimulated enough by oxygen lack and when the carbon dioxide has risen to the requisite minimum level. A form of periodic breathing is liable to set in until the oxygen and carbon dioxide tensions reach their normal levels.

Apnœa seldom occurs during open administration of ether; analysis of the air under such masks has shown 2% to 4% of carbon dioxide—adequate to stimulate breathing. The respiratory movements are not only an indication of the ventilation of the lungs, but also of the excitability of the nervous system and of the circulation and of the composition of the blood; they afford more varied information to the anæsthetist than an examination of the pulse.

The counterpart of apnœa is found in the difficult and laboured respiratory movements which constitute dyspnœa. The former, as we have seen, indicates the absence of stimulation; the latter is evidence of vigorous excitation of the nervous system.

Cyanosis depends on the absolute amount of reduced hæmoglobin in the blood, and will be present when anoxæmia is produced suddenly. Many factors modify it, such as increase or decrease in blood volume, so that it is not so evident after hæmorrhage or shock, and in any condition of anæmia in which the red corpuscles are less than normal. In nitrous oxide and oxygen anæsthesia a little cyanosis is generally disregarded. McKesson[9] states that in anæsthesia of normal depth plethoric persons may exhibit considerable cyanosis. Anoxæmia produced gradually does not necessarily give cyanosis.

Carbon dioxide looms largely in all the physicochemical reactions of the blood, and the formation of the acid H_2CO_3 and the base $NaHCO_3$ plays a great part in maintaining the normal pH value of the blood under all conditions. The respiratory centre is sensitive to variations of carbon dioxide at a tension as low as that of 1·6 millimetres of mercury, which corresponds to a scarcely measureable increase in the hydrogen ion concentration of the blood. When the hydrogen ion concentration tends to rise, pulmonary ventilation is increased, the level of the alveolar carbon dioxide tension is lowered, followed at once by lowered arterial carbon dioxide tension, that is, lower hydrogen ion concentration. There appears to be a specific action of carbon dioxide on the respiratory centre, apart from dissolving in the blood as H_2CO_3 and raising the number of hydrogen ions.

In the lungs, with the body at rest the venous blood with carbon dioxide tension of 46 millimetres of mercury is exposed to an alveolar air carbon dioxide pressure of 40 millimetres of mercury. Owing to the six millimetres of mercury difference of tension on the two sides of the membrane, carbon dioxide passes out of the blood into the lungs, enabling normally the appropriate amounts of carbon dioxide to be evolved.

Acapnia, the condition of lack of carbon dioxide in the blood, occurs in the states of apnœa and Cheyne-Stokes breathing. When carbon dioxide tension is too low to stimulate the respiratory centre, Nature generally applies her remedy of apnœa till carbon dioxide, continuously passing from the tissues to the blood, accumulates again.

The question of the relation of abnormal states of respiration to blood alkali is complicated. Carbon dioxide is carried mostly in the bicarbonates of the plasma; there is also a carbamino-fraction[10] existing in the red corpuscles. In 1925 Henderson[3] was inclined to view the lowering of blood alkali as a secondary result of acapnia, rather than as the result of an excessive production of acids. He now[11] uses the word acarbia to define an asphyxial diminution of the bicarbonates of the blood, and states that an alteration of pH of the blood really tells us, not whether the balance of acids and alkalis in the body is abnormal, but rather that the sensitivity of the respiratory centre is abnormally high or low.

> The control of the amount of alkali in use in the circulating blood by the pressure and amount of carbon dioxide in solution appears to be one of the fundamental biologic reactions in the living body. Oxygen lack is the initial cause and first stage of asphyxia, out of which develops the second stage, characterized by carbon dioxide lack and a compensatory decrease of the bicarbonates of the blood without considerable loss of alkali from the body.

Lack of carbon dioxide causes the hæmoglobin of the blood to hold on more tightly to its oxygen. An adequate carbon dioxide tension is necessary

for the oxygenation of the tissues, as shown in the curves of the dissociation of hæmoglobin. Henderson[11] has found that if carbon dioxide is inhaled after anæsthesia, the normal amount of alkali bicarbonate in the blood is quickly restored.

It has been claimed that animals, placed for prolonged periods in an atmosphere with an increased carbon dioxide tension, adjust their breathing to this new set of circumstances and, on being returned to a normal atmosphere, show alarming failure of respiration, often leading to death.

Uneven Lung Ventilation.

Professor Haldane[12] describes uneven lung ventilation as follows:

> In normal breathing of ordinary air the lung ventilation is so regulated that the partial pressure of carbon dioxide in the mixed alveolar air is maintained at a certain level characteristic for each person, usually corresponding to about 5½ per cent., while that of oxygen is about 14 per cent. The latter pressure, if it was evenly distributed in the alveoli, would be sufficient to saturate the hæmoglobin of the arterial blood to about 97 per cent. Actually, however, it is not evenly distributed, even with the breathing normal, so that some alveoli have more and some less oxygen. The consequence of this is that owing to the shape of the dissociation curve of oxyhæmoglobin the higher oxygen at some places does not compensate for the lower oxygen at others, and the mixed arterial blood has a lower oxygen pressure than the mixed alveolar air, even when the breathing is normal. Owing to the very different dissociation curve for carbon dioxide in blood, there is no corresponding difference for the carbon dioxide in the alveolar air and mixed arterial blood. When the breathing is shallow and rapid, even though the volume breathed per minute be much increased, the deficiency in the mixed arterial oxygen pressure becomes more exaggerated and easily causes symptoms of want of oxygen.

This has cumulative effects on the respiratory centre itself, the higher nervous centres, the heart and other organs.

Defective oxygenation from uneven ventilation may be avoided by: (i) adding oxygen to the inspired air, (ii) adding a little more carbon dioxide than is usual in the inspired air. When there is deficient oxygen intake there is present an abnormal stimulus to breathing, shallow and of increased rate, which washes out carbon dioxide.

Carbon Dioxide in Induction of Anæsthesia.

Two desired things in anæsthesia, on which I think most will agree, are, first, that depth should be maintained as evenly as possible, and, secondly, that the required depth should be attained as quickly as possible, the excitement stage being made practically non-existent. On the use of carbon dioxide in inducing anæsthesia I cannot do better than quote freely from Haggard.[13]

A gas or vapour dissolves in the blood according to the laws of gases; it remains in or comes out of solution according to the tension of the gas in the alveolar air. Blood at body temperature dissolves fifteen times as much ether as is in the air in contact with it when equilibrium is reached. The amount of gas going into solution in a liquid which has no chemical attraction for the gas depends on its solubility and is proportional to the partial pressure of the gas.

When blood containing ether is brought in contact with the tissues, the ether distributes itself through both media until an equilibrium of pressure is reached a second time. Some tissues have a greater affinity for ether than others; the lipoid tissue of the central nervous system is particularly affected.

For the new alveolar air at the end of each inspiration there is a fresh lot of blood in the lung capillaries that has come back from the tissues, ready to take up or give out ether, as the case may be.

The rate of absorption will depend upon the volume of the pulmonary circulation and the volume of respiration; the first increases along with the latter. Doubling the volume of respiration doubles the amount of ether brought into contact with the pulmonary blood. That is, two litres of air at a given concentration will bring as much ether into the blood as one litre at double the concentration.

The concentration of ether in the central nervous system is the determining factor in the anæsthetic action of whatever amount of ether is at the time in the body. As the central nervous system constitutes about 5% of the total body weight, the other 95% acts as a buffer and stabilizes the ether concentration in the central nervous system. Owing to its plentiful blood supply and affinity for the anæsthetic, the brain and spinal cord will approach saturation with ether at the tension in the arterial blood at a much more rapid rate than the rest of the body; so the more nearly the ether in the arterial blood can be brought to its anæsthetic concentration in its initial level, the more quickly will anæsthesia develop. To give a very high concentration is to be met with coughing, laryngeal spasm and increased flow of mucus and injury to the lung mucous membrane. By inducing a fourfold or fivefold increase in the volume of the air breathed, but with a low tension of ether, we can so quickly raise the ether tension in the arterial blood that unconsciousness is produced in a few breaths, and surgical anæsthesia in a few minutes. This increased breathing is easily produced by inhaling a little carbon dioxide with the anæsthetic-laden air.

In a few breaths it has reached the alveolar air, in a few more seconds it has reached the carotid sinus and respiratory centre. Its administration can be controlled according to its effect.

At the end of the operation, the greater the depth and hence the volume of respiration, the greater will be the amount of ether to leave the lungs at each expiration. At the same time, and as rapidly, ether leaves the blood for the alveolar air and leaves the tissues for the blood. Here again we can control breathing with carbon dioxide to produce de-etherization. After a short operation the ether concentration in the brain can be reduced sufficiently rapidly to have a semi-conscious patient in a few minutes. After a very long operation the de-etherization is best continued at intervals in the recovery ward, lowering the ether concentration in the blood and withdrawing it from the huge stores in the tissues.

In the days of Clover's inhaler inductions were quick. Sir Charles J. Symonds,[14] referring to a method he adopted in 1884, describes attaining surgical anæsthesia in five minutes. The expired air was rebreathed until the face became dusky, "by which time the child will be rendered so far unconscious as not to detect the admission of ether, which is now gradually admitted". This was anæsthesia by anoxæmia—rather hard on the central nervous system. Later he states:

> It requires some practice and judgement to know when to admit the ether and how long to continue before removing the face-piece and admitting air.

We might say there was in this more art than science. Such induction is a far cry from modern premedication, basal narcosis and nitrous oxide and oxygen administration; perhaps we are not of such stern stuff now.

Induction of Anæsthesia by High Percentage Carbon Dioxide.

I have not tried induction of anæsthesia by high percentages of carbon dioxide. It is described by C. N. Chipman[15] and H. A. Lakin.[16] When a concentration of 30% in oxygen was used, six to ten breaths seemed enough to produce unconsciousness, when ether was turned on. It could not have been ideally pleasant, as Lakin says it was not so successful in children because they did not cooperate.

Signs of an Overdose.

The excessive respirations become obvious; on withdrawing the carbon dioxide or decreasing the flow, overdose effects are rapidly abolished. When the respirations are very deep, I have noted that the face becomes reddened. Lakin,[16] experimenting on himself, mentions numbness when he inhaled 15% in oxygen. J. Ross Mackenzie[17] speaks of epileptiform convulsions, asphyxia, and rupture of alveoli from excessive intrapulmonary pressure in animal experiments. In anæsthetic work it is difficult to conceive of an overdose being given. It might occur when administration is left to nurses in a recovery ward.

Carbon Dioxide with the Open Method of Induction with Ether.

My experience with carbon dioxide to assist induction with ether has been pleasant. I was immediately impressed with its usefulness. I have used it in about 1,100 ether administrations, about half of the patients being children. To gauge the amount being used, I bubble the gas through water. With children I have been struck with the small quantity needed to give deep breathing, and the quickness of their response. In adults it is not so quick. I used to give first one or two cubic centimetres of ethyl chloride, and then I used to let the carbon dioxide flow as I started the ether; later I cut out the ethyl chloride altogether. With tonsillectomy cases at a children's hospital the constant repetition of the procedure gave me a good opportunity to compare cases and observe effects.

A mouth hook, such as is used for blowing in ether vapour, is a convenient thing with which to administer the carbon dioxide, as it stays in place under any open ether mask. It also keeps the teeth apart and makes any subsequent insertion of a mouth gag easy. The ether mask, scented with eau de Cologne or orange oil, is put on and a towel is folded to about six inches wide for children and wider for adults; the towel is wrapped around so as to form a vaporizing chamber above, with an opening at the top about two or three inches across.

Children.

I rarely find difficulty in coaxing the mouth hook into place in children. By the time the towel is arranged they find they have to breathe. One drop of ether is enough at first, then two, the rate of increase accelerating till the ether is given in nearly a constant stream. The carbon dioxide flow is adjusted to give the correct, steady, deep breathing; conscious efforts may mar it at first. Induction to the stage of ordinary surgical anæsthesia is quick—about three minutes in children, five in adults. I can testify to the uniform and speedy induction from a series of 426 children who had intratracheally administered ether. From commencing induction to the actual start of the operation the average time was seven and a half minutes. This time included carrying the child from one room to another, passing the catheter with a laryngoscope, fixing it to the mouth gag, adjusting the flow of ether vapour correctly, and sometimes waiting a minute or two for the surgeon. With a series having ethyl chloride and ether and no carbon dioxide the average time was nine and a half minutes.

I have questioned dozens of adults and older intelligent children, and from none have I received any remarks of finding the induction unpleasant. Of those who had had anæsthetics before, none compared it unfavourably. The common statement is that they only remembered taking one or two breaths. Such competent observers as sisters in the operating theatre have told me that I get smoother and quicker inductions than anæsthetists not using carbon dioxide.

Premedication.

As to premedication, children have had the appropriate dose of atropine only, generally by mouth; adults, morphine, 0·01 to 0·015 gramme (one-sixth to one-quarter of a grain), and atropine, 0·6 milligramme (one one-hundredth of a grain). Some had "Nembutal" and "Sodium amytal" when their use was fashionable. Latterly some children and adults have had "Evipan sodium" intravenously for induction. Here I have used carbon dioxide and ether cautiously, and have had no trouble in arriving at a satisfactory depth of ether anæsthesia.

During Maintenance.

I stop the carbon dioxide flow soon after induction is complete. The kind of semi-open mask used acts as an equalizer and prevents too sudden a change in the carbon dioxide content of the inspired air.

Special Inhalers.

Recently I have used a Dennis Brown ether inhaler in children; it has a high upper vaporizing chamber in which the carbon dioxide from the expired air is conserved. I have used it successfully without added carbon dioxide. (See Figure II.)

I have used a similar type of inhaler with adults, namely, Ogston's, which has a wire cage on top, raising the sides about six inches. (See Figure III.) Halton, of Liverpool, has suggested enclosing this with a section of motor car inner tube. (See Figure IV.) I tried this and found it very satisfactory, leading some carbon dioxide into the upper chamber to start good breathing.

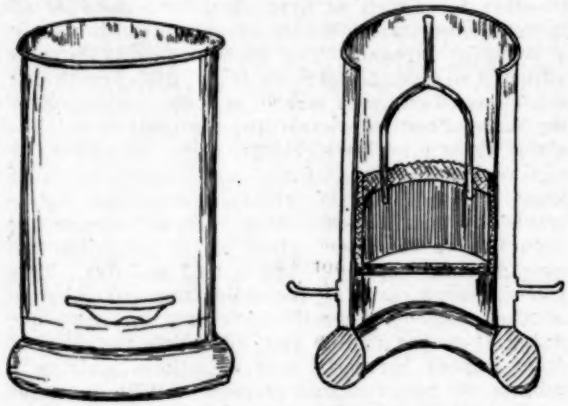

FIGURE II. Dennis Brown's ether inhaler.

The upper chamber in these masks forms, as it were, a modified rebreathing bag or extension of the lungs; there is a constant small stimulus to the respiratory centre. Halton has a tube fastened to the inside of the top part, through which oxygen or carbon dioxide can be delivered just above the gauze. The oxygen percentage inside will be lower than in air, but not too low for body requirements. These masks are certainly very economical in ether.

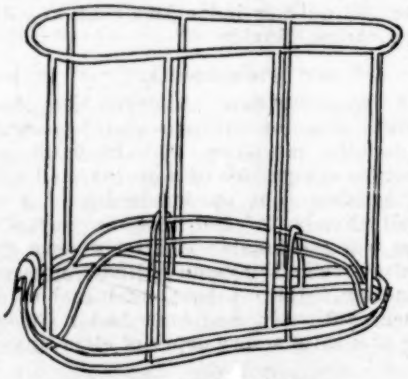

FIGURE III. Ogston's inhaler.

Induction with Ethyl Chloride.

There is no objection to using a little pleasantly scented ethyl chloride at the start, but I do not think that one gets the same smooth and quick induction as with ether alone.

Carbon Dioxide with Gas and Oxygen Anæsthesia.

Modern gas and oxygen machines have a valve to let in carbon dioxide in any desired proportion—a very desirable arrangement. I have not used one, but have had 10% "Carbogen" as an alternative to pure oxygen. In the rebreathing bag one can quickly build up a supply of carbon dioxide and, with variations in the proportions of the other two gases, get mixtures to suit the circumstances. I always start off with 10% "Carbogen" and may use it all through. It is my routine to do so with dental cases, even with a constant flow type of apparatus with rebreathing bag. The proportion of carbon dioxide does not appear to become excessive.

Carbon Dioxide Absorption.

Some arrangement to absorb carbon dioxide will probably be on all makes of gas apparatus in the future, T. A. B. Harris, who has designed one, mentions the possibility of anoxæmia from too low a tension of carbon dioxide in the alveolar air and blood, hindering oxyhæmoglobin giving off its oxygen. The ideal apparatus will have an adjustment so to retain as much carbon dioxide as is required.

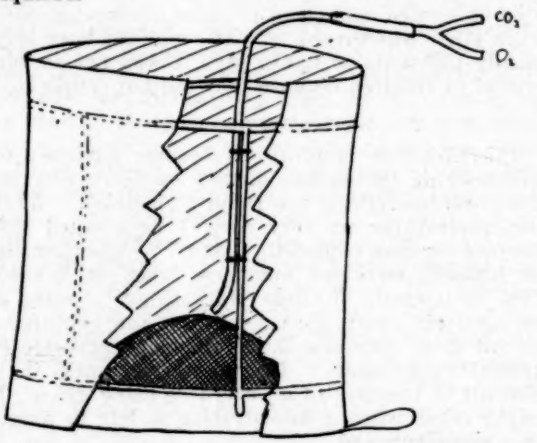

FIGURE IV.—Halton's modification of Ogston's inhaler.

Spinal Anæsthesia.

Seevers and Waters[18] point out that too little importance has been attached to the part played by the respiration in spinal anæsthesia. Owing to vasomotor nerve and skeletal muscle paralysis there are a decrease in the minute volume respiration figure, inadequate oxygenation of the blood and tissues with progressive loss of vascular tone over the whole body, fall of blood pressure, weaker heart action and less flow of blood through the lungs. This leads to anoxæmia of the respiratory and circulatory centres. The unevenly ventilated alveoli of Haldane will be present here. Raising the carbon dioxide tension in the alveolar air will do much to correct these departures from normal.

Carbon Dioxide in Shock.

We may define shock in an animal as the state which results from a fall of arterial pressure, which, if severe, may lead to death from oxygen want.

Professor McDowall[19] suggests that in the harmful centripetal impulses of Crile shock may result from loss of carbon dioxide due to over-ventilation, and that this variety of shock can occur in etherized persons. He has shown that in animals under chloralose over-ventilation does not bring about a fall of blood pressure, but that a fall occurs if the animal is etherized. Ether also appears to sensitize the blood vessels to the action of histamine. He thinks that acapnia may also be a primary factor in shock and that it cannot be ignored, for the evidence is complete that it reduces the activity of the vasomotor centre, by which the body attempts to compensate for a fall of blood pressure.

Where there is excessive breathing, producing acapnia, the anæsthetist may judiciously administer carbon dioxide to bring it under control and retain the alkali reserve. The shock I commonly see in upper abdominal operations comes on without quick breathing, though, as I often used intratracheal insufflation in these cases, washing out of carbon dioxide can go on unsuspected. In the established condition of shock, when so much of the blood is out of active circulation, we can, especially at the end of the operation, give the necessary stimulus to the respiratory centre to improve the oxygenation of the blood.

Infliction of shock is in the hands of the surgeon, and the best treatment is to prevent it. With a slow operator and much exploring and packing in of swabs an anæsthetist is hard put to it to steer his patient through to safety. A surgeon has said that no operation should last longer than one hour—a very good rule, especially when ether is the anæsthetic.

Post-Operative Depression.

Henderson has conducted an ardent and untiring campaign on the use of carbon dioxide in conditions of depression. He states that the influence of carbon dioxide on the circulation is as important as that which it exercises as a stimulant of the respiratory centre. He thinks that it combats the insufficiency of the venous circulation; this is the principal cause of the condition of post-operative depression, the analogy of which with the effects of hæmorrhage is evident. Restoring the carbon dioxide lost in the course of the anæsthesia and the operation relieves the acarbia, to use his latest definition of this condition. He says that there is no excess of carbon dioxide in post-operative depression, but an anoxæmia; the respiratory centre is too poisoned by the anæsthetic to respond and produce adequate oxygenation of the blood. The inactive centre promptly responds when enough carbon dioxide is given to reach the elevated threshold. As the oxygen supply is renewed by efficient deep respiration, the carbon dioxide threshold will drop to normal.

Post Operative Lung Complications.

Shock and upper abdominal operations lead us naturally to the subject of post-operative lung complications, in connexion with which they appear to be the principal causes.

A vast literature has grown up around these post-operative lung conditions. The pathological conditions are not always the same and are ascribed to various causes. R. J. S. McDowall[20] in 1923 drew attention to injury done to lung tissue by anæsthetics as a result of the actual contact with the vapour apart from concentration in the blood. This points to the desirability of avoiding a highly concentrated ether vapour.

V. E. Negus[21] calls attention to the action of the cilia and secretion of mucus in removing bacteria. H. A. Tarre,[22] by serial X ray photographs, has shown that there is an active peristaltic motion in expiration. Preexisting catarrh and irritation of ether causing an abnormal form of mucus to be secreted have been noted, as well as impurities in ether. All these causes point to the need for getting ether out of the body as soon as possible and freely ventilating the lungs. Then there is the massive collapse or atelectasis thought to be due to a plug of mucus blocking a bronchiole, or to a spasm of bronchiolar muscles in expiration, such as has been actually seen with lipiodol injections in the X ray screen by Pinchin and Norlock.[23] The plug of mucus by itself is not usually thought to be the whole cause. Sante[24] thinks that a simultaneous inhibition of the cough reflex by some toxic or reflex stimulus in association with an impairment of the respiratory muscles is necessary. The stimulus producing immobility of the diaphragm is probably the injured abdominal viscera, Nature's first thought being to guard them while healing takes place. But, as in good abdominal surgery the abdomen is closed with all lesions sutured, we need not trouble too much about the abdomen, but go ahead and see that the lungs function.

The Mucous Plug.

As to mucous plugs, I have one or two observations to make. At the Children's Hospital a lot of children have had bronchoscopic lavage for bronchiectasis. There are mucous plugs in plenty, stirred up by the stream of water which is then sucked out. It is inconceivable that every piece of thick mucus will be removed; some must lodge here and there in bronchioles. These children have a preexisting lung disease; they lie in a condition of respiratory and circulatory depression under "Avertin" for some hours, but do not get post-operative atelectasis or pneumonia. The cough reflex is reduced, but not abolished, for the time. The next morning they have returned to full normal activity, their circulatory and respiratory systems respond at a bound, as is normal in childhood.

Children after tonsil and adenoid operations have blood in their lungs, as has been abundantly proved by bronchoscopic examinations soon afterwards and by occasional *post mortem* examinations. They have plenty of potential bronchiolar plugs and have had ether, yet do not get post-operative lung complications, at least but extremely rarely.

Neither of these classes of patients have injured abdominal viscera to guard with their diaphragm nor painful and "splinted" abdominal muscles to

hinder breathing, and they return to normal life quickly. The additional factor mentioned by Sante is absent.

If one walks into a women patients' recovery ward, one sees the patients, especially if they have all had abdominal operations, propped up motionless in Fowler's position and staring forwards like statues of Queen Victoria. This leads me to say that there is a very good reason for the use of carbon dioxide, as by its stimulating effect on respiration and circulation, it is the only way we can artificially produce a condition approaching normality of the body functions. By early use of carbon dioxide after operation we hope so to aerate and expand the lungs that no small or large areas will become collapsed, and that the cilia and peristaltic movements will prevent mucus from stagnating anywhere. Administration for ten minutes by nasal catheter is the usual practice.

With an unobstructed airway there is really no need for oxygen with carbon dioxide; the air supplies plenty. There is a tendency to attribute magical benefits to "Carbogen". Pure carbon dioxide is so potent that, in the absence of a reliable apparatus made to deliver a definite amount per minute, someone should be alongside a helpless patient all the time. The oxygen in 10% "Carbogen" serves to dilute the carbon dioxide, so that any error is reduced one-tenth. My feeling is that, when it is administered by nurses in the recovery ward, not quite enough is given to produce the desired effect.

Bronchitis is often associated with a preexisting catarrh and, if looked for consistently, a mild form is said to be more common after ether than is suspected. According to modern views, at least in post-operative conditions, carbon dioxide will do more good by lung ventilation, encouraging the action of the cilia and the peristaltic movements of the bronchioles, than harm by spreading infection.

Carbon dioxide has been used in the recovery ward at the hospital for about eighteen months. I have no figures to give you of morbidity or mortality of post-operative lung complications for the institution, as such detailed statistics have not been kept. I see my own patients once or twice after operation, but only a keen resident medical officer who is in the wards daily can detect and appraise all chest signs and symptoms. From my records of 760 adults who had carbon dioxide induction and at least a little of the same gas afterwards, I find two deaths, that is, 0·25%. But both patients were bad operation risks, starting with pulse ratios outside the limits of good operation risk, and both had severe shock on the table. They serve very well to show the connexion between the two conditions.

Post-Operative Administration.

My own practice is at the end of the operation to give enough carbon dioxide through a nasal catheter when ether has been given by the open method to produce a well marked effect, aiming at deep, steady breathing. Just as in induction, I vary it according to the age and general condition of the patient. One is generally rushed on operation days at a public hospital, so that five minutes is the usual time that can be given. With intratracheal ether insufflation I let the gas into the stream of pure air flowing to the patient. With gas and oxygen I turn the dial to 100% oxygen and give a short burst of 10% "Carbogen". If I wish to give more, I switch to pure oxygen for a few breaths and then back to "Carbogen", using the same procedure with either a mask or intratracheal apparatus, until I see sufficient return of consciousness. I have not used carbon dioxide at the end of a spinal anæsthesia, as my patients have appeared to be in good condition, but I think it is indicated on general principles.

After "Avertin", Barbiturates and Morphine.

Carbon dioxide is recommended after "Avertin" and the barbiturates. Here it cannot assist in ridding the body of these drugs, as they are not volatile, but in cases of overdose the poisoned respiratory centre needs a higher tension than normal of carbon dioxide. Maintenance of good oxygenation of the tissues and raised blood pressure will keep the body going till the drug has been detoxicated. None of my "Avertin" or barbiturate anæsthesia patients have had carbon dioxide, except in the ordinary way after severe operations when post-operative depression was present.

Morphine poisons the respiratory centre, so that a high carbon dioxide tension is needed for it to function well.

Ether Convulsions.

Ether convulsions are not much heard of in Australia; I have never seen a case. The problem of how and why this interesting complication occurs has not yet been solved. Stoppage of movements has occurred after giving carbon dioxide. Its administration would seem to be indicated if we take into consideration the discovery of Lennox and Cobb, that in epileptic individuals the attacks were stopped by breathing high carbon dioxide mixtures.

Resuscitation.

We have in our expired air a good mixture for resuscitation, containing about 16% oxygen and 4% carbon dioxide. The old direct method of mouth-to-mouth insufflation has been handed down from the dim past. It was abandoned about the seventies because of faulty physiological reasoning about carbon dioxide. The method has been brought up several times in recent years. In an emergency cylinders may be empty, but the lungs of the bystanders are not. Remember that too efficient artificial respiration, especially if a stream of air or pure oxygen is being blown down the lungs, will remove carbon dioxide from the alveolar air and will make natural breathing difficult to start, because the carbon dioxide tension in the blood has become too low to stimulate an already depressed respiratory centre.

Other Methods of Administration than Inhalational.

C. Cot[25] and M. Loeper,[26] of France, are keen on other methods besides inhalational, namely, peritoneal, rectal, subcutaneous and transcutaneous

methods. Subcutaneous introduction is said to give a mild or gradual effect over a moderate period of time. Transcutaneous administration, that is, sitting in a mineral acid bath or in a cabinet containing pure carbon dioxide, is hardly suited to immediate post-operative therapy.

Some Advantages of Carbon Dioxide.

With carbon dioxide induction can be made quick and pleasant.

By shortening both induction and recovery of anæsthesia the actual duration is appreciably shortened and less harm is done to the central nervous system.

Varying depths of anæsthesia during a long operation may be quickly reached and removed by the use of carbon dioxide.

Acapnia, a cause of shock and post-operative depression, can be prevented with carbon dioxide.

The administration of carbon dioxide is the best prophylactic treatment of post-operative lung complications.

The administration of carbon dioxide may be the turning point to success in resuscitation.

References.

(1) Ogston: "Perhalation Method of Ether Administration", *The British Medical Journal*, May 13, 1922, page 762.
(2) A. Ley: *"Das Chloräthyl, ein äusserst brauchbares, aber mit Vorsicht anzuwendendes Mittel für den Rausch oder kürzere Narkosen"*, *Zentralblatt für Chirurgie*, October 15, 1921, page 1502.
(3) Y. Henderson: Quoted by M. S. Pembrey, *The British Medical Journal*, December 19, 1925, page 1180; also "Four Lectures on General Anæsthesia".
(4) E. Fischer: *"Die intraoperative und postoperative Kohlensäureinhalation, eine wertvolle Unterstützung der Inhalationsnarkose"*, *Zentralblatt für Gynäkologie*, Volume LII, August 11, 1928, page 2010.
(5) S. Wright: *Proceedings of the Royal Society of Medicine*, Volume XXVII, 1933-1934; "Applied Physiology", Fifth Edition, 1934, page 392 *et sequentes*.
(6) R. Gesell and C. Moyer: "Is Breathing Fundamentally a Reflex Phenomenon?" *Quarterly Journal of Experimental Physiology*, Volume XXV, April, 1935, page 13.
(7) McMechan: *Proceedings of the Royal Society of Medicine*, Volume XIX, 1925-1926, Section of Anæsthesia, page 38.
(8) J. S. Haldane and J. B. Priestley: "Respiration", 1935.
(9) McKesson: "Directions for Assembly and Care of the McKesson Indicating Nargraf", page 23.
(10) F. J. W. Roughton: "Carbon Dioxide Transport in Blood", *Physiological Reviews*, Volume XV, April, 1935, page 241.
(11) Y. Henderson: "Fundamentals of Asphyxia", *The Journal of the American Medical Association*, Volume CI, July 22, 1933, page 261.
(12) J. S. Haldane: Discussion on therapeutic administration of oxygen and carbon dioxide, *Proceedings of the Royal Society of Medicine*, Volume XXV, March, 1932, page 621.
(13) H. W. Haggard: "The Absorption, Distribution and Elimination of Anesthetics", *Current Researches in Anesthesia and Analgesia*, Volume V, February, 1926, page 1.
(14) C. J. Symonds: *The British Medical Journal*, September 8, 1928, page 465; "Use of Clover's Inhaler in Tonsillectomy and Removal of Adenoids", *ibidem*, September 29, 1928, page 581.
(15) C. N. Chipman: "A Report on the Use of Carbon Dioxide 25% and Oxygen 75% and its Effects on the Reduction in the Coagulation Time of the Blood", *Current Researches in Anesthesia and Analgesia*, August, 1926, page 200.
(16) H. A. Lakin: "Use of Carbon Dioxide during Induction and Maintenance of Ether Anesthesia", *Current Researches in Anesthesia and Analgesia*, March-April, 1928, page 85.
(17) J. R. Mackenzie: "Convulsions during Surgical Anæsthesia", *The British Medical Journal*, March 14, 1931, page 440.
(18) M. H. Seevers and R. M. Waters: "Respiratory and Circulatory Changes during Spinal Anesthesia", *The Journal of the American Medical Association*, September 17, 1932, page 961.
(19) R. J. S. McDowall: "Experimental Shock, with Special Reference to Anæsthesia (Arris and Gale Lecture)", *The British Medical Journal*, April 22, 1933, page 690.
(20) R. J. S. McDowall: "The Effect of Anæsthetics on the Lungs", *The British Medical Journal*, January 13, 1923, page 61.
(21) V. E. Negus: "Bronchoscopy in Post-Operative Lung Complications", *The British Medical Journal*, May 20, 1933, page 874.
(22) H. A. Jarre: *"Röntgenphysiologische Studien"*, *Zentralblatt für Chirurgie*, February 28, 1931, page 526.
(23) A. J. S. Pinchin and H. V. Morlock: "Acute Massive Collapse of Lung following Lipiodol Injection", *The British Medical Journal*, May 30, 1931, page 930.
(24) L. R. Sante: "Massive (Atelectatic) Collapse of Lung", *Annals of Surgery*, August, 1928, page 161.
(25) C. Cot: *"Carbogénothérapie et carbothérapie: l'utilisation de CO_2 en médicine générale"*, *La Presse Médicale*, December 17, 1932, page 1902.
(26) M. Loeper *et alii*: *"Avantages et inconvénients respectifs des diverses voies d'administration thérapeutique du gaz carbonique"*, *La Presse Médicale*, April 12, 1933, page 577.

ANÆSTHESIA IN AMERICA.[1]

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THREE very delightful and profitable months have recently been spent visiting the more important anæsthetic services in the United States and Canada. On every side I met with the utmost kindness and hospitality. On many occasions special methods were demonstrated in cases in which ordinary routine technique would otherwise have been used. Altogether I covered some 8,000 miles in the States and in Canada, and the centres visited were San Francisco and Los Angeles, Vancouver, the Mayo Clinic, Madison, and Chicago. The Atlantic City Congress then intervened and lasted for a week, and after that I went to Philadelphia, New York, Boston, Montreal and Toronto, Buffalo, Cleveland and Toledo.

Such a tour could of course only mean a bird's eye view so to speak of each centre, but perhaps one gets more of permanent value by adhering to such a plan than by concentrating on a particular centre and spending all or most of one's time there. The salutary effect of hearing the most varied opinions about one and the same technique or drug helped greatly to get a perspective, true or not, of the anæsthetic position today, as it is exemplified by American anæsthesia.

The Atlantic City Congress was a most striking assemblage. Some 8,000 doctors attended and it was a combined meeting of the American Medical Association and the Canadian Medical Association. The Anæsthetic Section numbered about 200 strong. England was represented by Sir Francis Shipway and Dr. Gillespie. The only shadow was the absence of Dr. McMechan owing to ill-health.

It is impossible and unnecessary to try to give you full details of all I saw, but I want to mention those recent advances either in methods or drugs which I think will interest you and endeavour to convey the impressions and conclusions I arrived at concerning them. I did not see chloroform given once. Ether was administered in relatively few cases as the sole method of anæsthesia, but I saw little of the nurse anæsthetist who doubtless uses it more frequently. Ethyl chloride is seldom used, mainly because efficient gas machines are practically always available for induction and so on. Endotracheal methods are not used so extensively as in this country, except in a few of the centres. I understand that the visit of Magill a few years ago gave a great fillip to this technique in America.

[1] Read at the annual meeting of the Australian Society of Anæsthetists, Melbourne, September, 1935.

Carbon Dioxide Absorption Methods.

The first thing that forcibly struck me was the large number of anæsthetists using carbon dioxide absorption methods in gas anæsthesia in the land where gases are infinitely cheaper than in this country. One must of course take into account the economic factor in the increasing use of the expensive gas cyclopropane; but, even allowing for this fact, some other reasons for its extensive use must be present. These are well summed up by Woodbridge[1] as follows:

1. The resulting quiet breathing with carbon dioxide absorption methods saves the energy output of the patient and in abdominal cases the surgeon's temper.

2. The resulting conservation of heat and moisture by the continuous rebreathing tends to counteract shock.

3. It is probable in this method that an increased percentage of oxygen can be used in the mixture.

4. Post-anæsthetic vomiting is diminished, particularly in thyreotoxicosis.

5. When using explosive mixtures the explosion hazard is decreased on account of the internal moisture in the circuit and the presence of a closed system.

6. Relatively small quantities of gases are used, hence tiny cylinders can be employed which greatly assist portability.

Several surgeons with whom I discussed anæsthesia and who were familiar with the surgical effect of this technique volunteered the information that they considered it, from their aspect, the greatest modern advance in anæsthesia.

The type of absorber is not important so long as it is efficient. The two most commonly in use are: (*a*) the to and fro absorber of Waters which fits on close to the mask, and (*b*) the circuit absorber manufactured by Foregger, which is attached to the gas machine itself. The former is much the cheaper owing to the absence of valves *et cetera*; the heat generated by it (never more than 47° C., even in the centre of the soda lime) does not cause irritation of mucous membranes by its proximity to the mask, but it certainly is more awkward to manipulate. Equally good results seem to be obtained with either by those experienced in their use.

The type of soda lime used in the absorber is important—Wilson's mesh, four to eight, is that recommended. It is not expensive and one filling of the canister will last for six to ten hours' anæsthesia.

The question one naturally asks is: "Does this depletion of carbon dioxide interfere with the hydrogen ion concentration in the blood?" The carbon dioxide content of air is 0·04%. Samples of the mixture taken from the bag in Waters's method and from the inspiratory tube in the circuit absorber show a carbon dioxide content averaging 0·2%. One can therefore assume that there is no starvation of carbon dioxide. As Woodbridge puts it: "The absorber effectively maintains a level of carbon dioxide below the point of marked physiological effect and yet safely above the zero point."

The carbon dioxide absorption technique would appear to be applicable to any type of gaseous general anæsthesia, with the exception of that administered through a nasal inhaler under pressure in oral and throat operations.

Cyclopropane.

The investigation of the clinical use of cyclopropane naturally occupied a fair amount of my time. I think you will be interested to hear the "low down", so to speak, of it.

Cyclopropane, an isomer of propylene, was first described by Freund in 1882 and was first experimentally used on animals by Lucas and Henderson,[2] of Canada, in 1929, who "rediscovered" it whilst investigating the impurities of propylene. It is a colourless, slightly pungent gas, has a specific gravity of 1·46, and it is distributed in cylinders at 75 pounds pressure to the square inch. It is almost odourless in percentages used clinically. It costs in America 45 cents a gallon.

It was first used on the human subject by Waters, of Madison, in 1930, and in that centre it has so far been given to some 4,000 patients. Guedel, of Los Angeles, has administered it to 1,600 patients. Paul Wood, of New York, in his paper at the Atlantic City Congress, deals with 800 cases. Nine hundred patients were anæsthetized with cyclopropane at the Lahey Clinic in Boston in 1934. Griffith, of Montreal, has given it 1,200 odd times; Bourne, of the same city, has given it many times, and so on in other centres throughout both countries; and they continue to use it.

What, then, are the advantages of cyclopropane over the other two commonly used gases, nitrous oxide and ethylene? First, large percentages of oxygen can be given with it in mixtures anæsthetically efficient. As Griffith[3] puts it: One has to get an altered conception of gas anæsthesia when using cyclopropane—it is added to the oxygen in quantities only sufficient to attain anæsthesia, whereas in using other gases one adds oxygen to the gas only in sufficient quantities to supply the minimum metabolic needs of the body. Narcosis has been produced with as low a concentration as 4% of cyclopropane in oxygen. Waters and Schmidt[4] have investigated the average percentages of cyclopropane necessary for various stages of surgical anæsthesia, and the figures are as follows:

Third stage:	First plane (roving eyeball) ..	7·4%
	Second plane (fixed eyeball) ..	13·1%
	Third plane (intercostal paralysis)	23·3%
Fourth stage	(respiratory arrest)	42·9%

When produced in the presence of high oxygen concentrations such respiratory arrest has not been found to cause harm, provided ample tidal exchange is artificially maintained.

The quiet, almost negligible, respiratory movements are a very definite feature of cyclopropane anæsthesia and require considerable getting used to.

There appear to be three main factors in their causation: (i) the gas is at no stage of anæsthesia a respiratory stimulant; (ii) the large percentage of oxygen used diminishes respiratory movements; (iii) the carbon dioxide absorption method, which is economically an essential adjunct, removes any tendency to stimulation of the respiratory centre from excess of carbon dioxide.

The quiet breathing, pushed if necessary to respiratory arrest, is utilized extensively in abdominal work. Guedel even goes to the extent of deliberately producing arrest in the early stages when he wishes to introduce an endotracheal tube. Although these manifestations are startling to those unused to this gas, the patient is at all times a good pink colour, owing to the amount of oxygen in the mixture; this in contradistinction to the appearance of the patient under the so-called secondary saturation with nitrous oxide. The closed system and bag make artificial respiration an easy performance, which is just as well, as it not infrequently has to be resorted to. Incidentally the term "artificial respiration" in this anæsthesia is being replaced by "passive respiration". Whether this is a salve to anæsthetic conscience or is used to avoid unduly perturbing the anxious surgeon I could never quite determine.

The second great advantage of cyclopropane is that it gives better relaxation than either of the two other gases commonly used.

The explosiveness of cyclopropane and oxygen is on a par with that of ethylene and oxygen, but the closed system and moistening of the mixture with total rebreathing has nullified this hazard. No account of an explosion has so far appeared.

A certain amount of evidence is now available regarding the toxic effect of cyclopropane on various organs. Bourne and Ruginsky,[5] of Montreal, report:

> Cyclopropane does not damage normal liver in dogs even after repeated administrations nor after long periods; it does not enhance the liver damage purposely produced by chloroform nor impede the usual recovery of the liver from chloroform poisoning even when cyclopropane anæsthesia is prolonged; and it does not cause liver impairment in starved dogs even after a three-hour period of anæsthesia.

There is further no evidence of renal damage from the use of this gas.

One now comes to its all-important effect on the cardio-vascular system, and at the present time the evidence is incomplete and to a certain extent conflicting.

Waters and Schmidt and others[4] report that, so far as blood chemistry is concerned, a hyperglycæmia (post-anæsthetic) and an increase in blood phosphorus are the only changes of note. Whilst increased capillary bleeding has been said by surgeons to be due to cyclopropane, Waters and his co-workers do not consider there is any real evidence to incriminate it. They report that *post mortem* examinations were performed in fifteen of forty deaths which followed in the post-operative period after cyclopropane was given, and the pathologist's findings on the myocardium were as follows:

> It has been difficult to evaluate the lesions found, as the patients had been on the whole extremely handicapped, not only by chronic lesions, but by acute terminal infections. Therefore any notation as to lesions must be accepted as subject to correction on more extended study.
>
> Heart: A definite tendency towards hyaline degeneration and necrosis of heart muscle fibres has been noted . . . the unusual character of the lesion suggests the possibility that it was due to cyclopropane, yet the later occurrence of it might discredit that suggestion.

Perhaps the most interesting work lately accomplished has been a series of electrocardiographic records taken during surgical anæsthesia under different anæsthetic agents, general and local, by Kurtz *et alii* at the Wisconsin General Hospital.[6] A paper on this subject was read at the Atlantic City Congress and I was privileged to peruse it carefully afterwards. It has not yet been published. Electrocardiographic records were taken in 109 cases during 113 operations. The ages of the patients varied from sixteen to seventy-three years. Of the patients, 69% had no demonstrable cardio-vascular lesions beforehand, 31% had cardiac abnormalities, definite arteriosclerosis or hypertension.

The anæsthetic agents used were as follows: cyclopropane, 41; ether, 20; "Procain", 13; ethylene, 11; nitrous oxide, 10; "Vinethene", 7; chloroform, 6; "Avertin", 5.

Cyclopropane was the anæsthetic of choice for many of the "poor risks", for example 23 out of 41 (hence in the majority with heart lesions).

Electrocardiograms were taken the night before, immediately before the operation, during induction, at intervals of one to five minutes throughout the operation, as the patient was coming out, ten hours afterwards, and in a few cases during convalescence. A Victor electrocardiograph was used. A colossal amount of work was entailed in the interpretation of the results, as you can imagine (25 to 60 tracings were taken during each operation). One of the points that stand out in the results is shown in Table I.

TABLE I.

Electrocardiograms taken Ten Hours after Operation compared with Controls taken before the Operation.

Anæsthetic Agent Used.	Identical Percentage.	Similar Percentage.	Different Percentage.
Cyclopropane	7	68	25
Ether		84	16
"Procain"	8	54	38
Ethylene		82	18
Nitrous oxide	10	60	30
"Vinethene"		86	14
Chloroform		100	
"Avertin"		80	20

Of all patients, only 20% maintained a normal rhythm throughout the operation. In all chloroform cases there was some type of disturbance. The "Procain" group showed comparative freedom from irregularities during the operation. Disturbances in rhythm were frequently noted before incision or during the preparation of the operative field.

Certain operative procedures appeared to increase the irregularities, namely, opening and closing the peritoneum, dragging on the gall-bladder and uterus, exploring the abdominal cavity by palpation, removing the appendix, and excising the thyreoid gland. The depth of anæsthesia did not appear to be important. No relationship was found between the irregularities and duration of anæsthesia.

The tendency was for the arrhythmia to advance as follows: first, occasional ventricular extrasystole; second, extrasystolic rhythm; third, runs of multiple extrasystoles from different foci; fourth, ectopic contractions with no normal beats. The normal was reached in the reverse fashion.

This final finding of ventricular extrasystoles from multiple foci was important and occurred in 10% of the cyclopropane group, and only in this group. This condition is alleged to precede ventricular fibrillation, and so is looked on by the investigators as a potential danger, and seriously for the present at least. Clinically any total irregularity of the pulse under cyclopropane, when accompanied by a rate of 100 or over, should be viewed with suspicion by the anæsthetist. Either ventricular extrasystoles from multiple foci or auricular fibrillation is present, if the heart has been beating regularly beforehand. Cyclopropane has produced ventricular fibrillation in dogs, but definite proof is lacking that this condition can be caused in man.

The cardiac disturbance is probably produced partly through the vagus nerve and partly by direct action on the heart muscle. Attention must again be directed to the fact that in this series of cases cyclopropane was used as the anæsthetic of choice in many of the bad risks. Nevertheless these arrhythmias of continuous ventricular extrasystoles due to multiple foci occurred only in the cyclopropane cases. The significance of this electrocardiographic finding is uncertain at the moment, and further work must be done along these lines before any conclusive result can be put forward. So much for this valuable, if at present somewhat inconclusive, piece of work.

What kind of results are the users of cyclopropane getting? Woodbridge and Sise, at the Lahey Clinic of Boston, told me that among their 900 cyclopropane administrations in 1934 they had two post-operative deaths, one from an air embolism, the other due to hæmorrhage from the pulmonary artery or vein. Both patients were very ill with lung conditions. Griffith,[3] in a report on his first 350 cyclopropane administrations, reports no pulmonary complications and three post-operative deaths. Peritonitis was found as the cause of death in each case on *post mortem* examination.

The best statistical investigation up to date is that of Waters and Schmidt.[7] They compare the post-operative morbidity among 2,200 patients who had cyclopropane with that of 2,200 who had either nitrous oxide, ethylene, or ether. The results are as follow: (i) Respiratory complications are diminished in the cyclopropane group. (ii) Circulatory complications are increased in the cyclopropane group, especially as regards fall of blood pressure and incidents of shock, but cyclopropane was often the anæsthetic of choice in cases giving evidence of circulatory handicap before operation, on account of the larger percentage of oxygen which could be used. (iii) the post-operative mortality in the cyclopropane group was 4·19%; in the other group it was 3·99%.

Guedel told me that in his series of 1,600 cyclopropane administrations there were three post-operative deaths: one from an atelectasis of the lung and the other two from thromboses.

Bourne[8] reports very favourably on its use in obstetrics (including Cæsarean section).

Technique of Administration.

Prenarcosis.—The chief argument in regard to administration seems to be whether one should use scopolamine or atropine with the morphine. In the light of the uncertain significance of the cardiac arrhythmias and the possibility of their being due, at any rate in part, to vagal action, it seems logical to include atropine for the moment in the pre-anæsthetic injection.

Paul Wood,[9] of New York, is using subbasal doses of "Avertin" (his method I will refer to briefly later) in combination with cyclopropane; and in spite of respiratory depression being a theoretical certainty, is getting extraordinarily good results.

Induction and Maintenance.—Details varied in different centres, but the fundamentals of induction and maintenance were the same. The rebreathing bag was first filled with oxygen and the mask was applied. Cyclopropane was then run in at 600 to 700 cubic centimetres per minute for anything from one-half to three minutes (according to the patient *et cetera*). The oxygen flow was then set at basal maintenance requirements, usually 300 to 600 cubic centimetres per minute. A pause was then observed until the patient's condition became stabilized, and further cyclopropane was added, if necessary, to attain the required depth of anæsthesia or to make up for leakage or lipoid absorption. It is a simple method, but, as you can imagine, requires experience to judge the dosage necessary. The question of whether one should start with the carbon dioxide absorber in the circuit is also the subject of argument. Waters, who is the exponent of using the absorber from the commencement, gets at least as good results as those who take the opposite view, so it probably does not matter which method is used. Waters lays stress on two golden rules: (i) beware of over-dosage, and (ii) always give plenty of oxygen.

Summing up, I have endeavoured to give you briefly the relevant points concerning cyclopropane from the evidence as it stands today, and you must form your own conclusions therefrom. One cannot see it in use without feeling that it is going to occupy a very definite place in anæsthesia and that it will encroach on the present field of nitrous oxide and ethylene. Waters, on whose capable

shoulders the bulk of the investigation has fallen, and whose baby cyclopropane is, so to speak, retains a perfectly open mind about it and welcomes criticism and argument. He will not commit himself at the moment and asks for another year of further clinical, pharmacological and experimental investigation before giving a definite opinion. He is doubtful at the moment as to how pure the present commercial product of cyclopropane actually is. Kurtz, in his paper, suggests that some of the cardiac irregularities found with the electrocardiograph may be due to propylene occurring as an impurity in the present commercial product. Waters also considers that the cardiographic findings require further elucidation. But even taking the foregoing into account, he is prepared at the present time, clinically, to give it as the anæsthetic of choice to the worst risk.

"Vinethene."

"Vinethene" is Merck's trade name for vinyl ether or divinyl oxide. It was first suggested as an anæsthetic agent by C. D. Leake, Professor of Pharmacology at the University of California Medical School. It decomposes when exposed to light and air, and should not be used more than twenty-four hours after the container has been opened. It is inflammable and its explosiveness is about the same as that of ethyl ether.

It produced anæsthesia with great rapidity when administered by either the open drop or modified closed method.

Its relative anæsthetic potency on dogs compared with ether is 4 to 1; with chloroform, 1·3 to 1·0.

The ratio of anæsthetic dose to lethal dose of ether and chloroform is 1·0 to 1·5; of "Vinethene" the ratio is 1·0 to 2·4.

Respiratory failure occurs before cardiac failure. "Vinethene" does not irritate the upper air passages, but increased salivation suggests the use of atropine as a preliminary injection.

The muscular relaxation that it produces equals that of ether. Recovery is rapid and is comparable with that from nitrous oxide. Nausea and vomiting are rare. Relaxation is alleged to be good with a roving eyeball stage of anæsthesia. Liver inefficiency is the only contraindication to its use.

In spite of the above somewhat glowing account, "Vinethene" was not used to any great extent at any of the centres I visited. Beach, at the Pennsylvania University Hospital in Philadelphia, has supervised its administration to some 5,000 patients and has probably had more experience with it than anyone else. During a very short visit to this centre I saw it used only for induction, by the ordinary drop method, as a preliminary to ether. The induction was quick and the patients appeared to have no discomfort. It is advised not to administer it continuously for longer than half an hour. Its odour has a suggestion of garlic.

It appears that indications for its use may be as follow: (i) As a substitute for ethyl chloride, both in induction before ether and for short periods of anæsthesia, in which ethyl chloride has been used alone in the past. It does not seem to have the same cumulative effect as ethyl chloride; relaxation is better and there may be less tendency to nausea and vomiting. (ii) As a substitute for ether in operations not lasting for longer than half an hour. It is more quickly eliminated than ether, and relaxation is alleged to be as good. (iii) As an adjuvant to gas administration, instead of ether, for the above reasons.

What its price will be in Australia I do not know. It seems, however, worthy of a trial under our local conditions, where so much ethyl chloride and ether are used.

"Thio-Nembutal."

The name "Thio-Nembutal" has tentatively been suggested by Lundy, of the Mayo Clinic, for a new barbiturate, similar to "Nembutal", except for a sulphur radicle attached to the urea side. It is used intravenously in a similar manner to "Evipan". About 300 patients had been given the drug at the Mayo Clinic at the time of my visit and the results were encouraging. Morphine, 0·015 gramme (one-sixth of a grain), and atropine, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), are injected as a routine prenarcotic, and after the solution of "ThioNembutal" is made up, about two to three cubic centimetres are given intravenously fairly rapidly. The needle is left in the vein and subsequent amounts of about half a cubic centimetre are injected when the patient shows signs of coming out, until the operation is completed.

It appears to be more effective in its action than "Evipan", relaxation is better, and jactitation in the induction stage, which sometimes occurs with "Evipan", appears to be absent. I saw several administrations for short operations, for example, excision of a lipoma *et cetera*, and they appeared to be entirely satisfactory.

Spinal Anæsthesia.

Perhaps in no other branch of anæsthesia, with the possible exception of prenarcosis, did I meet with such divergence of opinion in different centres as in regard to spinal anæsthesia. Drugs and methods which were lauded in one place were decried and condemned in the next. It would take a whole paper in itself to attempt to describe the multiplicity of opinions expressed and drugs and techniques used. The whole question of spinal anæsthesia is in the melting pot at the moment, and it appears to me that if one uses it and has a drug and method from which one is getting satisfactory results, it is wise to adhere to it until definite proof is forthcoming that other drugs and methods show superior advantages.

Although some anæsthetists are using "Percaine" when they want length of time with their spinal anæsthesia, the tendency is to rely more on "Pantocain" or mixtures of "Pantocain" and "Procain". Bourne is trying the Etherington Wilson technique with "Percaine", namely, injecting the hypobaric solution with the patient in the sitting position and

keeping this position for a time, varying according to the depth of anæsthesia desired; he reports good results. As a detail of technique Sise's fine gold needle would appear to give minimum trauma to the dura, but requires a guide inserted into the skin and subcutaneous tissues for its introduction.

Oxygen Therapy.

Oxygen therapy in the United States of America is largely in the hands of the anæsthetists. In this subject again there is much difference of opinion. Evans, of Buffalo, advocates a 100% oxygen atmosphere; others condemn this and say that it is unnecessary and even harmful. At the Atlantic City Congress a paper on the oro-pharyngeal method of oxygen insufflation was read and a very excellent film was shown; these came from the Wisconsin General Hospital. It is a simple catheter method, and the important point in it is the position of the tip of the catheter. If the tip remains in the naso-pharynx, only comparatively small percentages (20% to 30%) of oxygen are inhaled, no matter how fast the flow. If, however, the tip is in the oro-pharynx, then with a five litre per minute flow one can get up to 50%, and with the ten to eleven litre flow one can administer up to a 70% to 80% oxygen atmosphere to the patient. These percentages have been proved by bronchial analyses. The distance from the external nares to the oro-pharynx is roughly the external measurement from the nares to the tragus of the ear. Another method of determining that the tip is in the right position is to continue to push the catheter in until the swallowing reflex is excited and then to withdraw the tube slightly. Every twelve hours the catheter is removed and reinserted in the other nostril to avoid irritation. A humidifier is used to moisten and break up the oxygen. This can be home-made with little expenditure, so no expensive apparatus is required. The method can be used continuously without discomfort to the patient. The Wisconsin General Hospital is reticulated with oxygen pipes and no other technique except the above is practised.

Nurse Anæsthetists.

No account of American anæsthesia would be complete without some reference to the delicate subject of nurse anæsthetists. It is a phase of anæsthesia of which we in Australia are fortunately free. The startling fact was soon elicited that 97% of the anæsthetics in the United States of America are administered by nurse anæsthetists. The reasons of this, to us, almost incomprehensible position are as follows: (i) Until within the last few years the medical student received practically no instruction, theoretical or practical, in anæsthesia. Even a hospital intern or resident medical officer learned little or nothing of the subject. The result was that the average general practitioner started his professional life not knowing how, and in many cases not wanting to know how, to give an anæsthetic. (ii) The hospital authorities soon learned that to employ a nurse anæsthetist at $120 a month, instead of a qualified medical anæsthetist, was to their economic advantage. (iii) To supply the demand due to lack of trained medical anæsthetists, professional nurse anæsthetists have arisen, with their own strong association, who can automatically administer a moderately good routine anæsthetic of ether or even gas-oxygen-ether. Their knowledge of physiology, pharmacology, and pathology is, of course, lamentably lacking, but they do what they are told by the surgeon, who incidentally in many cases fails to recognize the advantages of modern anæsthesia. Such is the present position, and the sad fact remains that if tomorrow it was made illegal in America for nurses to administer anæsthetics, the supply of trained medical anæsthetists simply could not cope with the demand. The position is wisely being tackled from the foundation, and at the present time the best anæsthetic teaching centres are turning out a small, but steady, number of highly trained expert medical anæsthetists who are being distributed over the country to establish medical anæsthetic services, and the influence of these, it is felt, will gradually displace the nurse anæsthetists. The teaching of practical and theoretical anæsthesia to the student, the hospital intern and resident medical officer is now being carried out in a systematic manner in many centres, and this also, it is hoped, will aid in bringing about the desired end.

The statistical results of the clinics in which the anæsthetic services are in the hands of medical anæsthetists are proving to the anæsthetically ignorant profession the improved results from the medical as compared with the nurse anæsthetist. The States of Indiana and New York are endeavouring to hasten the metamorphosis by legislation, but one feels that the slower and surer method of educating both the profession and the public is more likely to attain the hoped-for result.

A great deal more of interest was seen, but time is lacking, and I must just briefly refer to one or two further aspects.

Wood's "Avertin" Technique.

Wood's[9] compact "Avertin" outfit and technique, as demonstrated by him at the Atlantic City Congress, are worthy of your notice. They do away largely with the argument of time expenditure and trouble necessary to make use of this valuable adjunct to general anæsthesia. The graduations on the bottle in the outfit were arrived at from figures collected in 1,200 cases. Groups of ten average surgical patients of like weight who were to undergo various operative procedures, were given varying doses of "Avertin". Using the minimum dose per pound of body weight at which all of the ten arrived at the operating theatre with sufficient sedation to permit the introduction of a pharyngeal airway without stimulating a reflex or arousing the patient, it was found that: (i) patients weighing 30 to 110 pounds required 80 milligrammes per kilogram; (ii) patients weighing 110 to 130 pounds required 76·5 milligrammes per kilogram; (iii) patients weighing 130 to 145 pounds required 75 milligrammes per kilogram; (iv) patients weighing

145 to 190 pounds required 70 milligrammes per kilogram; (v) patients weighing 190 to 220 pounds required 65 milligrammes per kilogram; and so the graduated scale on the bottle was worked out. Six and a half cubic centimetres of "Avertin" fluid is the maximum dose given, no matter how much the weight exceeds 220 pounds. For each ten years over fifty one-tenth of the scale dose is deducted, whereas with children, athletes, thyreotoxic patients and alcoholics one-tenth of the scale dose is added. In addition to the above dosage of "Avertin", Wood gives to the patient morphine, 0·015 gramme (one-sixth of a grain), and atropine, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), one and a half hours before the operation; otherwise the routine is the same as in other techniques. Ordinary tap water is used to make up the solution, and the Congo red testing liquid goes straight into the mixture in the bottle.

Guedel's Plug.

The final thing I want to refer to is Guedel's plug. Most of you probably are familiar with the inflatable Guedel-Waters cuffed catheter for completely blocking off the trachea in endotracheal anæsthesia. This plug of Guedel is made of soft rubber; it fits round the catheter like the inflatable cuff and serves the same purpose. Guedel claims that the catheter *plus* plug is easier to introduce into the trachea than the cuffed catheter, and is less likely to go out of order. It bears mostly on the aryepiglottic ring and also upon the ventricular folds and vocal cords, but to a lesser extent. It is practically self-retaining, does not have to be inflated and will withstand a pressure of air-tightness equal to that of the cuff. It will fit the average larynx of patients between the ages of eight and sixty years. It causes less irritation than the inflatable cuff, the nidus of which is lower in the trachea, and hence coughing is less likely to occur under light anæsthesia when the plug is used.

Conclusion.

The startling technique of secondary saturation with nitrous oxide-oxygen, as practised by the Toledo men, was seen, but to put forth the pros and cons of its use would require a paper in itself. Likewise the question of the relative merits of the various gas machines on the market is a huge and debatable subject. Suffice to say that the ideal gas machine for all purposes is not yet invented. All have their faults. The introduction of cyclopropane and the wider use of carbon dioxide absorption methods have altered our previous conception of what are physiologically the fundamentals of a good machine, and it is another subject that at the moment is definitely in the melting pot.

My tour was an experience I would not have missed. One cannot visit the various medical anæsthetic services in the United States of America without coming away stimulated and full of a profound admiration and respect for the excellent work being accomplished, sometimes in the face of what is at least passive resistance. And my gratitude is very great, not only for what they showed me, but for how they showed it.

References.

(1) P. D. Woodbridge: "Better Gas Anæsthesia: Carbon Dioxide Absorption Method", *New England Journal of Medicine*, March 23, 1933, page 632.
(2) G. H. W. Lucas and V. E. Henderson: "A New Anæsthetic Gas: Cyclopropane", *Canadian Medical Association Journal*, Volume XXI, 1929, pages 173 to 175.
(3) H. R. Griffith: "Cyclopropane Anæsthesia: Clinical Record of 350 Administrations", *Canadian Medical Association Journal*, August, 1934, page 31.
(4) R. M. Waters and E. R. Schmidt: "Cyclopropane Anesthesia", *The Journal of the American Medical Association*, September 29, 1934, page 975.
(5) W. Bourne and B. B. Ruginsky: "Effects of Cyclopropane on the Normal and Impaired Liver", *Canadian Medical Association Journal*, November, 1934, page 500.
(6) Paper read at the Atlantic City Congress on Electrocardiographic Studies during Surgical Anæsthesia, by Kurtz, Bennett and Shapiro, of the Wisconsin General Hospital (not yet published).
(7) R. M. Waters and E. R. Schmidt: "Post-Operative Morbidity in 2,200 Cyclopropane Cases compared with that in 2,200 of Nitrous Oxide, Ethylene, or Ether Cases", *Current Researches in Anesthesia and Analgesia*", January-February, 1935.
(8) W. Bourne: "Cyclopropane Anæsthesia in Obstetrics", *The Lancet*, July 7, 1934, page 20.
(9) Paper by Paul Wood at Atlantic City Congress, 1935 (not yet published).
(10) A. E. Guedel (Los Angeles): Personal communication.

THE CHOICE OF ANÆSTHESIA IN SOME SURGICAL CONDITIONS.[1]

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I SELECTED the subject of my address, hoping that my remarks would evoke discussion. The ideal in anæsthesia has not yet been evolved, and if I appear to be rather dogmatic at times, it is because I have been forced to arrive at decisions in particular cases in which I have been asked to anæsthetize a patient.

I believe that ether is undoubtedly the anæsthetic of choice for most surgical procedures, provided it is well administered. In this country, away from the capital cities, it is probably used in the vast majority of cases. Those using it are taught at our medical schools, and it is a pity that they are not better taught; they may receive the impression, as students or resident medical officers, that other anæsthetics are usually more suitable. Ether should never be used when the patient is a diabetic or when the patient has any infection of the respiratory tract.

I believe that gaseous anæsthetics are much more limited in their scope than I once thought. In this regard, too, the question of teaching arises. Under present conditions it is impossible to teach students or resident medical officers to be competent users of nitrous oxide or ethylene. The attempts of the unskilled to use these anæsthetics result in much more harm to the patient than would be occasioned if ether were used unskilfully.

It is very easy for us to be unintentional liars about the anæsthetic of choice, because we are apt to convince ourselves too easily. But I think it is to our credit that we should admit our mistakes and

[1] Read at the annual meeting of the Australian Society of Anæsthetists, Melbourne, September, 1935.

change our opinions accordingly. For some years I was quite convinced, and I did my best to convince surgeons, that nitrous oxide and ethylene were the only general anæsthetics that need ever be used, and that, perhaps combined with the use of "Novocain", they were applicable under all conditions. At present I am just as convinced that I was quite wrong, increasing experience having forced me to the conclusion that, with certain definite exceptions set out below, the ends of the patient are best served with a well given light ether anæsthetic, because, generally speaking, the surgeon is then able to operate with a minimum of trauma.

As a basis for discussion I set out my personal opinion on the choice of anæsthetic for certain types of operation: (i) oral surgery, including teeth extraction and tonsillectomy; (ii) surgery of the head and neck, including brain surgery, mastoid operations and thyreoidectomy; (iii) thoracic surgery, including thoracoplasty, lobectomy and hydatid disease; (iv) breast surgery; (v) abdominal surgery; (vi) perineal surgery.

Oral Surgery.

The first essential in oral surgery is to maintain an adequate airway, and the best way of doing this is to use a wide-bored catheter attached to a rebreathing bag, into which ether vapour is allowed to flow slowly from any of the usual intratracheal machines. The pharynx should be packed with gauze round the catheter to prevent blood or mucus from entering the trachea. If a machine is not available, excellent anæsthesia may be obtained by attaching the catheter to a two-way bottle, the glass tubing attached to the catheter stopping above the surface of the ether and the tube leading from the air being under the surface of the ether.

Teeth Extraction.—For teeth extraction, although nitrous oxide and oxygen given under pressure through a nasal mask are often satisfactory, it is sometimes difficult to obtain the requisite depth of anæsthesia; it requires practice and, as the apparatus is not always available, I believe that the use of ether with a wide-bored catheter, as detailed above, is the most satisfactory method.

Tonsillectomy.—For tonsillectomy a variation is necessary because the large catheter may hamper the surgeon. Here it is better to use a Davis-Boyle gag for the administration of ether. The patient should be placed in a position with the head extended and a sand-bag placed under his shoulders. A gauze pack in the naso-pharynx adequately prevents the entrance of blood into the retro-nasal space, and the position prevents blood entering the trachea. The amount of ether inhaled by the surgeon may be reduced by the use of a small bore catheter attached to an intratracheal machine, in conjunction with the Davis-Boyle gag.

Surgery of the Head and Neck.

I have not had enough experience with anæsthesia in the surgery of the brain to have reached any definite conclusions, and I look forward to the discussion to enable me to profit by the experiences of others. From my limited knowledge I would say that local anæsthesia was the most satisfactory, and, failing this, intratracheal insufflation of ether should be used. On the few occasions on which I have used gas I have been disappointed because there seemed to be an increase in hæmorrhage compared with that occurring when I had used the intratracheal method of giving ether.

Chronic Mastoid Operations.—In chronic mastoid conditions ether administered intratracheally either by insufflation or by inhalation is the best form of anæsthesia, because it removes the anæsthetist from the field of operation and allows him to watch for any injury to the facial nerve. The best method of doing this is to keep a finger on the angle of the mouth, for then the slightest stimulation of the nerve will give an impression to the finger very closely resembling that experienced when a fish takes the bait.

Acute Mastoid Conditions.—In acute mastoid conditions, because there is always an associated acute infection of the respiratory tract, ether should not be used. The gas anæsthetics are not suitable, because the bleeding is increased, and when, as is frequently the case, bronchitis or bronchopneumonia is present, cyanosis may be very troublesome. The best anæsthetic to use in these conditions is chloroform. Very little is needed and, because a very small mask or even a piece of folded gauze can be used, the anæsthetist can keep clear of the operative field. I believe that none of us is making sufficient use of this drug. A surgeon remarked to me recently that if chloroform had been newly introduced it would have been hailed as a very satisfactory anæsthetic in many conditions.

Thyreoidectomy.—In thyreoidectomy the choice of anæsthesia depends upon the presence or absence of toxæmia. There is no condition in surgery in which anæsthesia plays such an important part, and in many cases in which a patient who has had a toxic goitre removed has a stormy convalescence, the anæsthetist must take some share of the blame. Three conditions are always necessary for good anæsthesia: (i) a non-suffocative induction, (ii) a light anæsthesia with rapid elimination of the anæsthetic, (iii) an absence of the least trace of cyanosis. These criteria are more important in toxic goitre than in any other condition. I am quite sure that the best anæsthetic at our disposal for this condition is nitrous oxide and oxygen, the maximum amount of oxygen being used. It does not matter if the patient phonates throughout the operation. The use of this anæsthetic has done more to reduce the mortality rate in thyreoidectomy than in any other surgical condition; but even so, a badly given gas anæsthetic is more dangerous than a well-given light ether anæsthetic. With nitrous oxide and oxygen anæsthesia there may be some slight respiratory obstruction when the lobes of the gland are being delivered, but this is only temporary, and the intratracheal administration of gas or ether is never necessary and always harmful.

With non-toxic goitres nitrous oxide and oxygen anæsthesia is still the best anæsthetic to use, because the patients have a better convalescence, with, as a rule, little vomiting. But there is no strong objection to the use of ether as there is in toxic cases, and when the gland is very large and definite respiratory embarrassment is present before operation an intratracheal catheter should be used. The tracheitis which almost invariably follows the use of the catheter in toxic cases does not seem to occur so constantly in non-toxic cases, nor when it does is it usually followed by such ill-effects.

Thoracic Surgery.

Thoracoplasty.—I believe that the operation of thoracoplasty should be performed under regional anæsthesia. Occasionally a patient is too nervous for this to be done, in which case nitrous oxide and oxygen should be given as well as the local anæsthetic. Because the patient, when placed in a position suitable for the surgeon, has one lung out of action and the other compressed by the patient's weight, some degree of cyanosis is always present. This adds to the difficulties of the anæsthetist and increases the risk to the patient.

Lobectomy.—I have had no experience of anæsthesia for lobectomy and hope to hear from some of you who have.

Hydatid Disease of the Lung.—In hydatid disease of the lung a method of anæsthesia must be used whereby the lung may be inflated and the cyst made accessible for attachment to the chest wall after the pleura has been opened. This may best be obtained by giving ether intratracheally with a large catheter. The inhalation method is used until the pleura is to be opened and then the bag is discarded, the catheter is attached to the pump and the speed and pressure are so adjusted as to inflate the lung. When the hydatid is in the right lung, it is possible to pass the catheter into the right bronchus to inflate that lung only.

Amputation of the Breast.—With amputation of the breast there is always some shock when the breast is actually removed, and occasionally this may be very pronounced and even fatal. In milder cases recovery as a rule occurs before the patient leaves the table, but because of this very real risk of severe shock I believe that all breast amputations should be done under nitrous oxide and oxygen anæsthesia. I have never been worried during the administration of this anæsthetic in these cases, nor have I met with any deaths from operative shock, as I have on three occasions with ether.

Abdominal Operations.—In abdominal operations, except in the presence of diabetes or pulmonary disease, ether is the anæsthetic of choice, but there are four conditions in which I think that a variation may be advisable:

(*a*) In all operations on the stomach I am sure that the patient has a much better convalescence after anæsthesia with ethylene and oxygen than after ether. Provided that the surgeon can obtain all the relaxation and exposure that he requires, ethylene and oxygen should be used; but there should be no hesitation about changing over to ether if the surgeon's needs are not met, for nothing is more irritating to the surgeon than to be told that the patient will "soon be softer", and nothing is more harmful to the patient than to have an eleven-stone surgeon pulling hard on his abdominal wall.

(*b*) Cholecystectomy is usually performed with less harm to the patient under ether anæsthesia, but when jaundice is present ethylene should be used. Chloroform, of course, and, to a less extent, ether, are definitely toxic to liver cells, whereas ethylene, if given with sufficient oxygen, is not.

(*c*) Acute pancreatitis is always accompanied by shock and toxæmia. The patients take ether badly and the best anæsthetic to use is ethylene and oxygen. Infiltration of the abdominal muscles with 1% "Novocain" solution will insure adequate relaxation and avoid the necessity of using ether.

(*d*) When prostatectomy consists of a simple enucleation following previous suprapubic drainage of the bladder, I am sure that the best interests of the patient are served by the use of ethylene and oxygen. For patients undergoing an operation of the open type necessitating the Trendelenburg position, intratracheally administered ether is the best type of anæsthetic to use. If pulmonary complications preclude the use of ether, a low spinal anæsthesia is most satisfactory and may be induced with 1·5 cubic centimetres of an 8% solution of "Novocain" or with 10 centigrammes of solid "Novocain" dissolved in cerebro-spinal fluid. With this dosage the fall in blood pressure is negligible if 1·0 cubic centimetre of ephedrine is given just before the spinal injection.

For perineal operations the ideal anæsthetic is that known as caudal block, using 30 to 40 cubic centimetres of a 2% "Novocain" solution injected into the caudal canal. The only objections to this type of anæsthesia are: (i) that in a very small percentage of cases no anæsthesia occurs and then a low spinal anæsthesia has to be used, and (ii) that there is a delay of from about twenty minutes to half an hour from the time of injection until full anæsthesia is induced.

Conclusion.

I regret that this paper has been so discursive, but, as I said at the beginning, I wished it to form the basis of a discussion. I have not mentioned prenarcosis because I think this part of the subject merits a separate discussion. My conclusions as to the choice of anæsthetic for different types of operations have been arrived at only after experiencing difficulty at the time of anæsthetization, causing surgeons difficulty in the satisfactory performance of their work, and watching patients have troublesome after-effects from the anæsthetic. My ideas therefore are based on practical experience and do not involve theoretical considerations. I do not imagine that you will all agree with my views, and I am quite open to conviction on any of them.

ANÆSTHETIC FAILURES.[1]

By AN ANONYMOUS CONTRIBUTOR.

MANY years ago at a medical dinner a learned judge, the guest of honour, made a remark that has remained in my memory ever since. Replying to the toast of his health, he said: "You members of the medical profession, gentlemen, are in a very favoured position—the world acclaims your successes and flowers cover your failures."

It is to some of my flower-covered failures that I wish to refer today. In all, nine patients have died upon the operating table whilst I have been administering an anæsthetic to them. I had intended to divide these deaths into two classes, the unpreventible and the preventible, or the acts of God and those of myself. On second thoughts perhaps it would be better just to report the cases and allow you to place the blame where it properly belongs.

The first patient, then, was a man who was suspected of having an abscess of the lung. He was anæsthetized by the ethyl chloride and open ether sequence. An exploratory puncture was made into the lung substance and very shortly afterwards the patient became greatly cyanosed and, in spite of all efforts, died, apparently from asphyxia. A *post mortem* examination showed that the needle had punctured a large vein communicating with the abscess cavity and that both bronchi were full of blood, the patient having died from drowning.

The second of these patients was a woman, aged fifty years, who had a toxic goitre. I must apologize for the scantiness of this history, but the only notes available are those from public hospital records.

The patient is reported as complaining of dyspnœa and œdema, where I do not know. Pulmonary and aortic murmurs were present, and the apex beat was in the middle of the axilla. The pulse rate was 130 per minute and her systolic blood pressure was 130 millimetres of mercury. The patient was anæsthetized with ether by the open method, and an intratracheal catheter was introduced. At this time gas and oxygen were not available at the hospital. After the first lobe of the thyreoid had been removed, I informed the surgeon that the pulse was uncountable and that the systolic pressure had dropped to 70 millimetres of mercury, and asked him to complete his operation as soon as possible. In spite of my protests, he insisted on going on, and the patient died whilst the skin sutures were being inserted.

My errors in this case were twofold: first, the wrong anæsthetic was used, although it was the best available at the time, and secondly, I should have insisted on the surgeon stopping when I considered the patient was in danger.

The third patient was a child, aged eight years. On the previous day tonsils and post-nasal adenoids had been removed. Hæmorrhage occurred and a dose of "Hæmostatic serum" was given. Hæmorrhage recurred twice, and on each occasion a further anæsthetic was administered and also a dose of serum. On the following day I was asked to give another anæsthetic to enable the surgeon to remove a plug from the right tonsillar fossa. I had put only one or two drops of ether on the mask when the child ceased breathing. On the coroner's advice a *post mortem* examination was made, which showed that the child had died from pulmonary thrombosis.

In this case considerable pressure was brought to bear in order to prevent me from reporting the death, but I have always been glad I resisted the temptation.

The fourth case was that of a man, aged fifty-five years, ill for two months following a tooth extraction. An exploratory puncture of the lung was to have been done. Had I seen the patient beforehand, I should certainly have refused to give him a general anæsthetic, as his respirations were 80 per minute, his pulse rate was approximately 220 per minute, and he appeared to me to be moribund. However, I weakly consented to give him ether by the open method, and after a few minutes he expired. *Post mortem* examination showed gangrene of both lungs, and there was apparently no healthy lung tissue left.

My mistake here was, of course, in consenting to give the patient an anæsthetic at all.

The fifth case was that of a stoutly-built man of forty years, suffering from a large post-nasal growth. When seen for the first time in hospital he was frightfully cyanosed. His colour could be compared only with that of a child with congenital *morbus cordis*. I asked the surgeon to perform a preliminary tracheotomy under local anæsthesia, as it was obviously not a safe case for a general anæsthetic, unless an airway were provided below the obstruction. The patient, however, absolutely refused to have anything done without a general anæsthetic and assured us that he would return home to die rather than be operated on whilst conscious. He had previously come down to the city for treatment, but had returned home for this same reason. Rather than let him do this, I consented to anæsthetize him. Immediately I poured a few drops of ethyl chloride on the mask spasm occurred. We forcibly opened his jaw and attempted to pass an intratracheal catheter, but the introducer broke under the strain. On account of his strong teeth, big tongue and thick neck, an attempt to pass a catheter blindly was unsuccessful. A rapid tracheotomy was performed, but too late. (Since that time I have always carried a second introducer and have never needed it.) *Post mortem* examination showed an easily removable non-malignant tumour, completely blocking both sides of the nose, but not coming below the soft palate. The pathologist was at a loss as to why the case had been reported as a death under anæsthesia, as there was no smell of anæsthetic in the body. The cause of death reported was spasm of the glottis.

Again in this case I should have refused to give an anæsthetic in the circumstances, and possibly the patient might later have allowed us to do a preliminary tracheotomy and his life would have been saved.

In the sixth case I will first give you the surgeon's story and then my own notes made at the time.

The surgeon reports:

> The patient is a woman aged seventy-one years. The diagnosis is an acute inflammatory condition of the right side of the abdomen, probably of the gall-bladder. Operation is to be done when the acute attack subsides. Five days later: The patient has had much pain in the last two nights and a probable cardiac attack two days ago. Operation was performed on the sixth day under ethylene and oxygen anæsthesia. Kocher's incision was made and the abdomen opened. The gall-bladder was tightly distended and full of pus. This was withdrawn with a syringe; it was at first turbid and later thick, with no bile at all. No stone was present in the common bile duct or cystic duct. The patient was not very fit and I asked the anæsthetist whether he thought she was good enough for a cholecystectomy. He thought she was. However, after seeing the fibrotic condition of the liver, and the gall-bladder

[1] Read at the annual meeting of the Australian Society of Anæsthetists, Melbourne, September, 1935.

well adherent to it, I decided against anything radical. I was then putting a tube into the gall-bladder when the anæsthetist reported that the heart had stopped.

And now the same patient from my own point of view. She was a woman, aged seventy-one, with myocardial degeneration and empyema of the gall-bladder. Her systolic blood pressure was 110 millimetres of mercury and her diastolic pressure 90 millimetres. She had been kept for five days in hospital in the hope of improvement, but, as she was getting worse, operation was decided on. Ethylene and oxygen were administered and her colour was bad all the time, although her pulse rate did not markedly increase, nor did her blood pressure fall. After twenty-five minutes, and just after I had told the surgeon that I thought she would stand a cholecystectomy, her heart suddenly stopped.

This was undoubtedly a cardiac and not a respiratory failure. I have always had an uneasy feeling that this patient may not have died on the table if I had given a bigger percentage of oxygen during her anæsthesia.

The seventh case was also that of an elderly woman with a diseased gall-bladder. Her age was seventy-five years and she had been ill for six weeks. Owing to a history of cardiac weakness, her physician did not think any operative treatment was advisable. An electrocardiogram had been taken, but unfortunately this has been mislaid, together with her history. An operation was rather suddenly decided on, and I was asked to give her a general anæsthetic. Her blood pressure prior to operation was: systolic 90 millimetres of mercury, diastolic 70 millimetres. Ethylene and oxygen were administered, and all seemed to be going well when suddenly, as in the previous case, the heart stopped.

I feel confident that these two patients should have been given nitrous oxide rather than ethylene.

The eighth case was of very great interest.

The patient was a heavily built man, sixty years of age, suffering from a gangrenous appendix. He was not seen prior to the time of operation, but on cursory examination made on the table he had a systolic bruit at the apex. No cardiac enlargement or irregularity was present. There was a vague history of a sudden attack of pain in the chest on the day before operation, but not sufficiently severe to require advice. The anæsthetic used was ethyl chloride, followed by ether given by the open method. After five minutes the skin incision was made and the heart immediately and dramatically stopped. The surgeon, noticing the cessation of breathing, looked at me inquiringly. As I lifted the mask, I said: "He probably feels something and is only holding his breath. You can go on; he is all right." The surgeon said: "If he is all right, why does he look like that?" By this time the mask had been lifted and it was plain that the patient was dead. The *post mortem* examination showed calcified and thickened coronary arteries and a calcified area in the heart muscle. Too late, I was able to get a detailed history, including the following, from a well known cardiologist:

I examined this man four years before the date of the operation. He stated that two months previously, whilst walking to catch a train, he experienced a pain across the chest, which was severe enough to make him cease walking. The pain then eased, but recurred with further walking. Some time later he could only walk about ten yards before pain came on. The pain was mostly across the front of the chest, substernal, sometimes in the left subcostal region or about the left shoulder. It was associated with a certain amount of tightness in the chest. The pain came on more readily when walking after breakfast, and was also likely to occur when walking against a cold wind. For the previous three weeks the pulse was intermittent about every third or fourth beat. He did not complain of shortness of breath or of indigestion. There was no difficulty with urination or nocturia. He smoked about four ounces of tobacco a week and drank on the average one gin per day. His past history included typhoid fever at the age of thirty-two, and epidemic influenza at the age of forty-six. He had been treated for nervous depression six years previously, but soon recovered. He was usually "nervy" and highly-strung, and had much commercial and domestic worry. His father was still living at ninety years of age; his mother died when aged fifty-six years. Examination showed a man of florid complexion and good nutrition. Frequent extrasystoles were present. Electrocardiograms showed extrasystoles that arose from various foci in the auricles, auriculo-ventricular node and right ventricle. The curves were of low voltage. *T*I was flattened, *T*II and *T*III were upright. The apex beat was about four inches from the mid-line. The heart sounds were clear. The systolic blood pressure was 150 millimetres of mercury, the diastolic 90 millimetres. The lungs and abdomen seemed normal. The urine was free from albumin and sugar. A report was sent to his medical attendant that the patient undoubtedly suffered from *angina pectoris*; in addition, from multiple extrasystoles, which were indicative of widespread irritability of the myocardium. The prognosis was regarded as bad. The advice included instructions for great care for an indefinite period.

This was the patient to whom I light-heartedly agreed to give a general anæsthetic after a cursory examination on the table! However, as the patient was a burly, fat man, gas anæsthesia would have been difficult. Spinal anæsthesia would have been more dangerous, and I suppose that the result would have been the same in any case.

The ninth case was that of a woman, aged thirty-five years and married for eight years. The surgeon reports:

She was seen twelve days ago by her own doctor because of free bleeding *per vaginam*, with clots and some pain. An enlarged uterus was present and a threatened or incomplete abortion was diagnosed. The menses had been regular until two months before, one period having been missed. The cervix was slightly dilated, with an offensive discharge *per vaginam*. The temperature was 101° F. The breasts were slightly active. Dilatation and curettage were attempted without success and the cervix packed. Three days later another operation was performed and a large uterine cavity was found, but no evidence of pregnancy. A fibroid was then diagnosed. The patient continued to run a temperature of from 101° to 103° F., had an offensive discharge, and was not improving. She was accordingly sent down to the city by car, a distance of nearly two hundred miles. On arrival, twelve days after her illness began, she was in a very exhausted condition. Her pulse was rapid, her temperature 101° F. There was a large, round, hard and tender swelling occupying the lower abdomen and pelvic cavity and extending nearly to the level of the umbilicus. The cervix showed evidence of having been recently torn by the tenaculum. She was diagnosed as having an infected or degenerated fibroid and was immediately sent to hospital. Next day the temperature at 9 a.m. was 105·4° F. and the pulse rate 160 per minute. At 1 p.m. the temperature was 101·6° F. and the pulse rate 136 per minute. The general condition did not show any sign of improvement and it was considered that operation afforded the only chance of recovery. At 5.30 p.m. hysterectomy and double salpingo-oophorectomy was done and a gauze drain inserted into the vagina. The patient's condition got worse on the table and, in spite of rapid closure of the abdomen, the patient did not recover. On incision of the uterus, it showed a large fibroid undergoing red degeneration.

When I first saw this patient prior to operation her pulse rate was 200 per minute and her respiration rate 80 per minute. She had a drawn, anxious expression and was obviously desperately ill. Her systolic blood pressure was 95 millimetres of mercury, her diastolic pressure 70 millimetres. The type of anæsthetic to be used was very seriously discussed and, as any operation must necessarily be of short duration, it was decided to use ether with oxygen, so as to enable the surgeon to do his work with the greatest ease and rapidity. In all, only twenty minutes elapsed between the first incision and the skin sutures, but during the whole time her condition got steadily and progressively worse until her death.

In this case I consider we anticipated Nature only by a few hours. Possibly I should have used nitrous oxide and oxygen instead of ether, but I really do not know.

These are the cases I wished to present to you. I am not proud of them, as you can well imagine, but possibly the reporting and discussion of them may be of service in preventing others from making similar mistakes.

Reviews.

ANÆSTHESIA.

IN his preface to "The Theory and Practice of Anæsthesia"[1] Dr. M. D. Nosworthy has laid down a fundamental principle of mastering the older before dabbling with the newer methods. This is excellent advice, in view of the tendency of the modern inexperienced anæsthetist to employ the more advanced methods, such as basal narcosis. Of this the author has written: "Basal narcosis must not be regarded as a short cut to proficiency; without a sound knowledge of the basic principles of anæsthesia its use is definitely dangerous."

It is refreshing to read that the author has still given a place to chloroform in the field of anæsthesia and has advocated teaching of its uses. He has wisely remarked "that to be afraid of using chloroform is almost as bad as being over-confident".

In a short but good account of the use of carbon dioxide a timely warning has been sounded against its use for a patient whose air passages are filled with mucus. The risk of plugging the smaller air passages is increased.

The chapter on shock has been well written from a practical viewpoint. The effects of light and deep anæsthesia and of the various anæsthetic agents have been well discussed in their relationship to the production of this condition.

Dr. Nosworthy has dealt with the difficulties of pure nitrous oxide and oxygen anæsthesia (for example, the tendency to the production of excessive anoxæmia) and the advantages to be gained by the addition of minimal quantities of ether or chloroform. In Graves's disease he has recommended the addition of minimal quantities of ether, any disadvantage from its toxic action being outweighed by its advantages. Many anæsthetists will agree with this.

Of the secondary saturation technique of McKesson he has not approved. It is interesting to note that in describing the machines typifying the various principles employed in gas anæsthesia mention has not been made of the McKesson Nargraf.

In the chapter on spinal anæsthesia, "Percaine" and "Stovaine" are the only drugs the use of which has been described. "Novocain", although mentioned, was not considered. The chapter is otherwise good.

Naturally, in a small book there must be omissions. The drop method of using ethyl chloride has been omitted. This is safer than squirting a few cubic centimetres on to the mask and awaiting developments. In view of the author's opinion, and an excellent one, that the character of the respiration is the surest sign in estimating the depth of anæsthesia, it would have been better to have enlarged upon the various types of breathing met with in each successive stage of anæsthesia. The section dealing with obstetrical anæsthesia could have been amplified. Nevertheless the author has performed his task well. The book, slightly over two hundred pages, is printed in large type, easy to read, and is full of references. The student, the recently qualified and the advanced anæsthetist will learn much from its contents.

[1] "The Theory and Practice of Anæsthesia", by M. D. Nosworthy, M.A., M.D., B.Ch., with a foreword by I. W. Magill, M.B., B.Ch.; 1935. London: Hutchinson and Company. Crown 8vo., pp. 223. Price: 12s. 6d. net.

MEDICINE FOR NURSES.

SEVERAL books have been published recently dealing with medicine as it concerns nurses. W. Gordon Sears has produced one of these, a compact volume, which in its 400 pages covers considerably more than any nurse need know of medicine and all that she may need to look up for reference purposes.[1] There is a distinct tendency in the literature of the nursing profession to supply text-books which set forth and clarify the theory of the more important divisions of medical and surgical knowledge and to leave the practical applications of these subjects to the tutor sisters and other senior members of the nursing staffs of hospitals whose function is to make nurses. In this way the pupil nurse is gradually being regarded educationally as a kind of medical student; this trend is not being criticized here, it is merely pointed out, though all nurses who use these books must understand that the books can supply only the theoretical background. Dr. Sears has compiled a thoroughly useful book; it is up to date and reliable and clearly written. Here and there we might question details. Can the small beat of the pulse due to the extrasystole always be felt, or can *pulsus alternans* be recognized at all by nurses? How far should we teach pathology to nurses; for example, need we describe the changes in the pneumonic lung in detail while giving lung abscess only brief mention? Incidentally the treatment of the latter is not generally surgical as is stated here. And is it necessary to describe the physical examination of the heart? These questions are more provocative than destructive, for there is no doubt that nurses need to have access to all manner of what should be to them interesting details about the maladies of their patients. A few subjects might be amplified, for example, the handling of the so-called cardiac asthmatic (whose nocturnal attacks are very much in the sphere of the nurse as regards prevention and treatment), and oxygen and "Carbogen" administration with an appliance such as the Davies-Gilchrist set.

This book may be recommended confidently as a useful reference text-book for nurses. It is a handy size and clearly printed, though here and there a slightly different selection of the type for headings might assist the eye.

[1] "Medicine for Nurses", by W. G. Sears, M.D., M.R.C.P.; 1935. London: Edward Arnold and Company. Crown 8vo., pp. 419, with illustrations. Price: 8s. 6d. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 21, 1935.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

ANÆSTHETISTS AND LOCAL ANÆSTHESIA.

PRACTICALLY the whole of the space in this issue is devoted to a report of the first annual general meeting of the Australian Society of Anæsthetists. On a previous occasion the objects of the society were set out in these pages; and the report of the first meeting shows that the society is doing its best to attain most of its objects. In due course no doubt some attempt will be made to organize or at least to encourage research into questions pertaining to anæsthesia; it would hardly be fair to expect anything to be done in this direction at so early a stage in the life of the society. Since anæsthesia enters largely into the practice of many practitioners, the papers and discussions will be read with interest. A high standard has been attained. Perhaps special mention may be made of Dr. Ivan Maxwell's discussion of some of the biochemical aspects of anæsthesia and of Dr. B. L. Stanton's paper on the pharmacological effect of some of the impurities in commercial anæsthetics. Important as papers dealing with the anæsthetic technique and the several anæsthetic agents undoubtedly are, contributions such as those of Dr. Maxwell and Dr. Stanton deal with fundamentals and, it is to be hoped, will be studied by all readers. In these two papers may be found several subjects in which members of the Society of Anæsthetists might undertake research. Through an unfortunate misunderstanding an important paper by Dr. Geoffrey Kaye, dealing with deaths from anæsthesia, has not been included.

None of the papers read at the meeting dealt directly with local anæsthesia. The cynical, not realizing the truth of the matter, might suggest that the omission was intentional. They might say that local anæsthesia is the province of the surgeon and not of the anæsthetist. The latter view would not necessarily be correct. More and more are anæsthetists becoming masters of the art of producing local anæsthesia, or local analgesia as it should be called. It is well that it should be so. The surgeon has quite enough to do in performing the operation. Field block and nerve block may well be carried out by the anæsthetist; and the same may be said of spinal anæsthesia. Moreover, these procedures are often associated with basal narcosis, and they may have to be supplemented in certain circumstances by gaseous or ordinary inhalation anæsthesia. An anæsthetist has to be present; he should be entrusted with the whole of the anæsthesia.

Local analgesia is coming more and more into use. It has several advantages over other forms of anæsthesia. If we except spinal anæsthesia, we may say that it is free from shock; it is safer than ordinary anæsthesia, and, if used skilfully, it is often more pleasant. Some enthusiasts use it extensively; their very enthusiasm may suggest to the uninitiated an exaggeration of the truth. Those, however, who have had experience of its use in their own bodies, or who have seen it used by a master of the art, will agree that the time is coming when surgeons and anæsthetists will have to adopt it, because patients will ask for it, if for no other reason. There is another reason why local analgesia will be more widely used in the future in Australia—

that the surgeon isolated from his fellow practitioners can by its means often carry out alone procedures which would otherwise be impossible. This aspect is really outside the present discussion, the object of which is to show that an anæsthetist, if he would be complete master of his craft, must be able to produce local analgesia if called upon by a surgeon to do so. A warning for those who would acquire proficiency in local analgesia is necessary; it has been well emphasized by Dr. C. E. Corlette in a contribution to *The Journal of the College of Surgeons of Australasia* of July, 1929. One who would begin to learn the art of local analgesia must begin at the beginning, not at the middle or the end. To begin with complicated procedures will be to court disaster, and to discredit an indispensable method.

Current Comment.

SERUM LIPOIDS IN DIABETES.

THE human body is a complicated affair. Efforts to investigate its functions are apt to render the problem of regulating them more difficult instead of simplifying it. Recently diets high in carbohydrate and poor in fat have been widely advocated for diabetic patients, and their use has been attended by considerable success. But, as so often happens, this form of treatment is much more to be commended than the theory on which it is based. As arguments for it, it has been claimed that hypercholesterolæmia is of grave prognosis in diabetes and that high fat diets may induce hyperlipæmia. Especial virtue has been ascribed to the determination of blood or serum cholesterol as a measure of the severity of diabetes, and as a criterion of the suitability of the patient's diet. Certainly it would be extremely satisfactory to have such a rational basis for the high carbohydrate/low fat dietary treatment of diabetes, but unfortunately the facts do not support these arguments. The truth is that we still know very little about the blood lipoids, and it is certainly impossible to make categorical statements about their relation to the severity of diabetes. It is quite as impossible to relate either cholesterolæmia or lipæmia to fat intake.

Evelyn B. Man and John P. Peters[1] record the results of an investigation undertaken to determine the incidence of hypercholesterolæmia in diabetes, to study the relation of the cholesterol level to that of fatty acids and phospholipoids in blood serum, and also to learn whether diets moderately high in fat give rise to hyperlipæmia. They examined the serum of 79 diabetic patients (20 male and 59 females of all ages) for cholesterol, lipoid phosphorus, and fatty acids. The venous blood used in the determination was withdrawn before the morning insulin or breakfast was taken. In all, 130 determinations were made on 79 patients. Serum proteins and blood sugar were determined simultaneously. No studies which were made when the patients were dehydrated, acidotic, pregnant, or in the puerperium were included. Of the 79 patients, serum cholesterol was normal in 42, above normal in 28, and below normal in 9. The normal range was considered to be 150 to 256 milligrammes *per centum*, though the authors remark that an attempt is being made to ascertain whether the range for females is the same as for males. Of the 28 patients with hypercholesterolæmia, all were females except two with liver cirrhosis, one with nephrosis, and one with vasomotor instability, although about 27% of the patients studied were men.

The low incidence of hypercholesterolæmia in this series undoubtedly depends on the fact that no diabetics with dehydration or acidosis were included. In diabetic acidosis a relative or absolute hypercholesterolæmia is regularly observed, which diminishes or disappears with the acidosis. In a previous investigation Man and Peters have shown that dehydration is largely responsible for the hypercholesterolæmia of diabetic acidosis. The capillaries are impermeable to both serum proteins and cholesterol, and hæmoconcentration causes both these constituents to increase. Omitting such dehydrated patients from their series, Man and Peters found that the level of serum cholesterol did not appear to be related to the severity of the diabetes, the fat in the diet, or the degree of arteriosclerosis. Severe hypercholesterolæmia was frequently referable to complicating conditions. It was found also in a group of patients who presented instability of the vasomotor reactions and carbohydrate metabolism. Mild hypercholesterolæmia was observed in obese females, but appeared to be related to the pattern of obesity rather than to the diet. A low blood cholesterol content was associated with malnutrition and lowered blood protein.

It has been stated that the level of the blood cholesterol may be taken as a criterion of the total blood fat. This matter requires further investigation. In this series of estimations the statistical correlation between lipoid phosphorus and cholesterol in blood serum was quite close. The correlation with serum fatty acids was far more irregular, but gross changes in the one were reflected in the others. It is not surprising, when one considers the differences in constitution and properties of fatty acids and cholesterol, that in spite of the general correlation between these two substances, considerable variations are observed. Such dissimilar substances are almost certainly metabolized by different processes. There is no reason why variations in blood cholesterol should

[1] *Journal of Clinical Investigation*, September, 1935.

correspond to variations in fatty acids. For example, an increase in serum fatty acids after food is not accompanied by a rise in serum cholesterol. Similarly the increase in fatty acids in diabetic acidosis far exceeds the increase in cholesterol.

Many conflicting statements have been made about the relationship between cholesterolæmia and arteriosclerosis. On this account an attempt was made to evaluate the degree of vascular degeneration in each patient of this series. Lipoid values and blood pressure were also compared. It was found that severe arteriosclerosis, with or without hypertension, was evident in patients with low, normal and high blood cholesterol.

The impression these authors derive from their study is that abnormalities of serum lipoids are related loosely, if at all, to diabetes. A high blood fat and a raised blood cholesterol are not a regular part of the diabetic syndrome, and when they do occur cannot be connected with the inherent severity of the disease. If hyperlipæmia or hypercholesterolæmia occurs in a treated diabetic it would seem necessary to seek the cause in peculiar features of the individual disease, or in a temporary acidosis due, for instance, to some infection. In a certain proportion, adequate explanations for hyperlipæmia can be found in associated diseases, such as cirrhosis of the liver, nephrosis, or other renal disorders. Man and Peters found that when these cases were eliminated, most of the remainder with greatly elevated lipoids fell into a group that presented one common feature, which may be roughly spoken of as instability of the autonomic nervous system. This was reflected also in instability of carbohydrate tolerance, which baffled regulation of insulin and diet. They found it impossible to define the syndrome further. They remark that the basal metabolism was frequently above normal. They are, of course, unable to resist the temptation to place the onus of the whole syndrome on the pituitary, that highly responsible gland, adding, however, the reservation that all this must remain mere speculation until the effects of hypophysis and mid-brain on lipoid metabolism are established.

THE STUDY OF VENOUS BLOOD PRESSURE.

CONGESTIVE heart failure when well established is so easy of diagnosis as to be obvious. Lesser degrees need careful examination, for though the characteristic signs may be present to some extent, they may not be objectively or subjectively obvious at first. The breathlessness and peripheral œdema creep upon the patient, the enlargement of the liver, the pulmonary œdema, and the cyanosis may be slight in degree, and the general venous engorgement may at first escape notice. It is with the last mentioned sign that we are here concerned. Lewis has drawn attention to its great importance, and has shown how reliable and how significant a sign it is, both in diagnosis and prognosis. It is not difficult to study filling of the veins at the bedside, and if it is desired to make this an accurate study it can be done by the use of very simple apparatus. W. B. Wartman has made exact determinations of the venous blood pressure in over 200 patients suffering from various diseases and his results are of some interest.[1] He employed a direct method in which a needle was inserted into an appropriate vein, usually the antecubital, and saline solution was allowed to run in from a manometer tube. The point of equilibrium could easily be read off. When possible the patient lay on his back with the vein level with the position of the right auricle; when this could not be done, as in the case of patients with failing hearts, the patient was allowed to sit up and the arm was brought up to the correct level. Thirty-one patients who presented no signs of circulatory embarrassment were used as controls; in these subjects the venous pressure varied between 60 and 120 millimetres of water, the average being 83 millimetres. Charts are presented in Wartman's paper illustrating several points, for instance, the frequency of high venous pressure readings in the subjects of congestive heart failure of arteriosclerotic or rheumatic origin; and the persistent rise in venous pressures in those patients whose circulatory failure was progressive. One interesting record shows how a patient with mitral stenosis and congestive failure improved with coincident fall in the venous blood pressure, but when a recurrence of the failure occurred later the pressure did not fall correspondingly and the patient died two months later.

The failure of the patient's venous circulation to adjust itself to approximately normal conditions could thus be measured, and some accurate idea of prognosis obtained. It was found, of course, that a high venous pressure did not always mean a declared cardiac failure, but cases in which the venous blood pressure readings were high could be taken as being potential cases of failure. When the pressure rose over 250 millimetres of water, it was found that the mortality rate rose steeply in a series of patients suffering from varied complaints. This might be expected, of course, but measurement, when it is accurate and free from fallacy, is always more convincing than generalization. Of this series of patients it was found that 69% died within three months, whereas of those whose pressure was below the critical level of 250 millimetres, only 8% succumbed.

Probably not many observers will trouble to record the venous pressures of patients, even though this could be done in hospitals while obtaining blood samples for routine tests. But is should be remembered that venous pressures are of supreme importance in cardiac disease, and perhaps the making of such estimations and their use to check the value of simple clinical observations might enhance the value of the latter and sharpen the acumen of the physician.

[1] *The American Journal of the Medical Sciences*, October, 1935.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Generalized Pustular Psoriasis.

T. N. GRAHAM (*Archives of Dermatology and Syphilology*, August, 1935) reports a case of generalized pustular psoriasis with typical psoriatic patches during and between pustular attacks. The patient presented also acute arthritis during pustular attacks, with resulting articular destruction. In a history of three attacks an increase in the number of typical psoriatic lesions, the appearance of numerous pustular lesions and acute arthritis have been concomitant. All the nails were involved and presented the typical features of psoriasis. It is stated that the question of the pustules being sterile or containing staphylococci is no longer considered of importance.

Unusual Cutaneous Symptoms Associated with Retention of Arsenic.

B. THRONE AND C. N. MYERS (*Archives of Dermatology and Syphilology*, August, 1935) report five cases of retention of arsenic, together with the laboratory findings. Four of the patients presented unusual lesions of the skin of a follicular lichenoid appearance. In acutely inflamed stages these lesions show minute vesicles at their apices and may show serous exudation. In stages of quiescence the vesicles are not present and the follicular lesions are hard. In all patients an extremely large amount of arsenic was found. It was considered that the retention of arsenic was the chief cause of the manifestations presented. The treatment of conditions due to arsenic is discussed. The significance of melaninuria in one case was not understood. It was suggested that conditions of this type, if not treated by eliminative measures, might undergo a transition to some form of lymphogranulomatosis. Apart from arsenical medication, occupational contacts and contamination of food must be kept in mind.

Calcinosis.

M. BOLAM (*The British Journal of Dermatology and Syphilis*, August-September, 1935) discusses the various types of calcinosis from a dermatological standpoint, with the report of a case of *calcinosis universalis* of metabolic type. Observations extending over the last two years showed little change in the levels of calcium and phosphorus in the blood, but the blood phosphatase level was found to be slightly raised. In two years the size of the calcified masses as determined clinically have not changed. The general resistance of the patient appeared to be lowered by a restriction of milk and butter in the dietary and by the taking of sodium dihydrogen phosphate. Radiological findings were normal for the bones, but many calcareous deposits were seen in the subcutaneous tissues of the legs. An analysis of this material showed a fairly high degree of calcification to be present. Microscopic examination showed that the calcification was limited to the subcutaneous fat, the corium being free. The histological picture suggested that in this case the calcification followed some degenerative changes in the fat lobules.

Actinomycosis of the Hand.

R. S. HOLLINGSWORTH (*The Journal of the American Medical Association*, October 19, 1935) reports a case of actinomycosis of the hand occurring in a man aged sixty-five years. The patient had been seen by several medical practitioners, who made a diagnosis of malignant disease and advised amputation. The lesion appeared two and a half years before the patient was seen by the author, and followed an accident when the patient ran a thorn into the back of his hand while he was cutting a hedge. Shortly after this accident the patient sought advice on account of a swelling at the site of injury. The swelling was cauterized. When the author saw the patient, the lesion consisted of numerous raised, ulcerated, pinkish nodules, containing foul-smelling, purulent material. At the site of the original injury there was a large ulcerated area; this involved the bases of the fourth and fifth fingers and extended onto the palm of the hand. The author found on close inquiry that the patient had at the time of his original injury treated a cow suffering from lumpy jaw. Branching mycelia were found. The condition was treated by excision, the raw area being painted subsequently with tincture of iodine and dressed alternately with a compound solution of iodine and 1% copper sulphate solution. For three weeks the patient received twice a week intravenous injections of 20 cubic centimetres of sodium iodide, and daily doses of dipropyl iodine proteinate by mouth. The end result was satisfactory.

Granuloma Annulare.

J. T. INGRAM (*The British Journal of Dermatology and Syphilis*, August-September, 1935) reports twelve cases of *granuloma annulare*, three associated with *lupus vulgaris*, and information has been collected from 261 cases reported in the literature. Two hundred and thirty-three cases of *lupus vulgaris* have been reviewed for the purpose of comparison. *Granuloma annulare* is undoubtedly a blood-borne toxic manifestation of a chronic nature. It is suggested that the chronicity of the lesions, the age of incidence of the affection, the frequency of a personal and family history of tuberculosis, especially when these figures are compared with those of a known tuberculous reaction of the skin, as *lupus vulgaris*, favour the view that *granuloma annulare* is itself a toxic tuberculous manifestation. The Mantoux test appears to give a positive reaction in the majority of cases, while guinea-pig inoculation has invariably yielded no evidence of tuberculosis. The presence of œdema is stressed as a histological feature and as determining the clinical characteristics of *granuloma annulare*.

Lichen Sclerosus et Atrophicus (Hallopeau).

FORTUNATO A. DIASIO (*The Urologic and Cutaneous Review*, October, 1935) reports a case of *lichen sclerosus et atrophicus*. This condition, described by Hallopeau in 1887, is a distinct clinical entity characterized by an irregular or angular, flat, shining, white papule, having on its surface dark, horny plugs or minute depressions and showing a tendency to grouping or confluence. The author reports a case that occurred in a girl, aged twelve years. He reproduces a photomicrograph of a section of the lesion showing atrophy of the epidermis, follicular plugging, fragmentation of fibres in the upper part of the cutis, and round cell infiltration in its deeper layers. He concludes that *lichen sclerosus et atrophicus* is distinct from *lichen planus* with atrophy and may readily be distinguished from the latter on clinical grounds, and in borderline cases by biopsy. Cardlike scleroderma (Unna) and other types of localized scleroderma are similar histologically to *lichen sclerosus et atrophicus*, and must be differentiated chiefly on clinical grounds.

UROLOGY.

Treatment of the Cord Bladder.

For the type of cord bladder in which tone is well maintained in both bladder wall and sphincter, E. L. Pierson (*The New England Journal of Medicine*, July 11, 1935) advocates transurethral resection of the internal sphincter. The continued effort of the bladder to force out urine through an uncooperative sphincter ultimately produces the same result as an enlarged prostate or contracted bladder neck. The precise neurological diagnosis is not of great importance in the selection of suitable cases. For this purpose it is well to make a cystometrogram to demonstrate the presence of sufficient tonus in the bladder wall. Then a cystogram, taken during the voiding of the opaque fluid, will clearly demonstrate the obstruction caused by the internal sphincter, if such obstruction exists. The antero-posterior view is the most satisfactory. It is essential that the external sphincter be shown to be efficient before operation is undertaken. After the usual preoperative preparation a small segment is excised from the posterior and

postero-lateral aspects of the internal sphincter with the resectoscope. By this simple procedure the risks of repeated catheterization or the inconvenience of suprapubic cystostomy are avoided.

Resection of the Nervi Erigentes in Painful Cystitis.

M. V. RICHER (*Journal d'Urologie*, January, 1935) states that the question of vesical sensibility is very complex for the physiologist. The surgeon, however, should not confuse himself too much with experimental results, often contradictory, but should rest on the sure ground of clinical observation and daily experience in practice. The best way to anæsthetize the bladder, without doing either too little or too much, is to perform sacral epidural anæsthesia. There are, therefore, nerve strands which lead vesical sensations of pain to the roots of the pelvic plexus. Division of the nerves from the second, third and fourth sacral segments will relieve pain by inducing anæsthesia of the bladder, but retention of urine results from unbalanced action of the hypogastric plexuses. This undesirable result may be neutralized by adding resection of the postero-superior cornua of the hypogastric plexuses as well as section of each hypogastric nerve. A gamma-shaped incision in the posterior peritoneum exposes the presacral plexus and each hypogastric nerve. After the operation a catheter is tied in for two days, after which it will be found that the patient can urinate without pain.

Surgical Treatment of Hydronephrosis.

IN discussing the treatment of hydronephrosis, the following points are emphasized by J. C. Kimbrough (*Journal of Urology*, February, 1935): (1) Selection of cases. Plastic surgery for the relief of hydronephrosis is only palliative and some degree of morbidity frequently persists. It is unsuitable for patients who insist on a rapid and complete cure. When the other kidney is sound, plastic surgery is unsuitable for a badly damaged kidney, but pathological conditions causing hydronephrosis are frequently bilateral, although of different degrees of severity. Conservative surgery is imperative in marked hydronephrosis of a solitary kidney, but nephrectomy is the operation of choice in elderly patients with fair function on the opposite side. (2) Technique. As regards the size of the pelvis requiring resection, no invariable rule can be given. Large, thin, atrophic pelves and thick, inelastic types do not tend to diminish in size with palliative treatment. Pelves with a capacity of 150 cubic centimetres or more usually require resection. Post-operative drainage of the pelvis is imperative after plastic work, and this is most satisfactorily obtained by nephrostomy. Splinting of the ureter is achieved by passing the ureteric catheter through the nephrostomy tube. The kidney should be fixed in its proper position at operation to prevent angulation of the ureter. Renal sympathectomy is optional. (3) Post-operative care. The ureteral catheter splint is removed in four to six days; the nephrostomy tube after three weeks. Perirenal drainage is maintained for six to eight days. The foot of the bed is elevated for seven days, the patient lies flat during the second week, and sits up in the third week. Late treatment consists in ureteral dilatation and pelvic lavage to prevent stricture formation and infection.

Recurrent Renal Calculi.

G. ILLYÉS (*Journal d'Urologie*, November, 1934) has made a study of problems involved in the recurrence of renal calculi involving a material of 842 surgical interventions. In 49 cases recurrence occurred, that is, in 8·3% of all cases. In order to avoid pseudo-récurrence, that is, calculi left behind at operation, very careful pre-operative localization by radiography and, if necessary, radiography of the delivered kidney, are advisable. After complete and careful removal of the kidney, careful hæmostasis and good drainage are necessary. True recurrence is due to three factors: (i) Infection. Most important of all are staphylococci, which render the urine alkaline and precipitate phosphates. After operation lavages of the renal pelvis and internal antiseptics should be administered. (ii) Retention. If at operation the renal pelvis is found to be very dilated, it may be reduced in size by a plastic operation. The problem is more difficult when the calyces are dilated and have rigid walls; in such cases, when infection is well established, nephrectomy is the only treatment of value. (iii) Stone-forming diathesis. This may become manifest at a certain period of life, but by itself is insufficient to determine the formation or recurrence of calculi; a local factor is necessary in addition. The particular diathesis may be influenced to a certain degree by appropriate hygiene and diet.

Tidal Drainage of the Urinary Bladder.

D. MUNRO AND J. HAHN (*New England Journal of Medicine*, February 7, 1935) describe an apparatus for tidal drainage of the urinary bladder. The objective appears to be alternate dilatation and relaxation of the bladder wall rather than decompression of the distended bladder or intermittent irrigation. A typical cycle is said to occupy about two hours. The bladder, being empty, gradually fills by the influx of urine and irrigating fluid until the intravesical pressure reaches a pre-arranged level, when the apparatus automatically siphons off the whole of the bladder content and the cycle recommences. Bladders suiable for tidal drainage are pre-eminently those associated with spinal cord injuries. "Cord" bladders are neither "paralysed" nor "automatic". Their degree of activity is a direct function of the degree of interference with the reflex response to a given intravesical stimulus. Four groups are therefore separable: (i) "Atonic cord bladder" during the period of spinal shock is due to the total inhibition of the reflex and is characterized by atonia of the detrusor. It is the optimum time for the institution of tidal drainage. (ii) "Autonomic cord bladder" follows as shock passes off. Reflex activity returns in the form of inefficient detrusor contractions, which open the internal sphincter but cannot expel the contents. A leaky bladder results. (iii) "Hypertonic cord bladder" is the condition arising when the sacral segments are destroyed, and is commonly known as the "automatic" bladder. (iv) "Uninhibited reflex cord bladder" occurs with a transection above the sacral segments. It is a normal bladder deprived of the inhibition of higher centres, and leads to incomplete evacuation of the bladder, which frequently passes unrecognized and leads to infection. The bladder wall may then revert to the atonic state. Tidal drainage prevents the accumulation of residual urine and prevents retrogression.

The Clinical Value of Cystometry.

M. MUSCHAT (*Journal of Urology*, April, 1935) limits the value of the cystometer for clinical purposes to a determination of the condition of the bladder wall. He states that a cystoscopist of very extensive experience may be able to detect a neurogenic bladder with certainty by inspection alone. The average urologist requires further assistance to be definite on this point, and the cystometer provides the necessary aid. The author's instrument is much simplified from the original design of Rose, and consists of a reservoir, a Wolfe bottle, a manometer, a three-way cock and appropriate tubing. The bladder is filled by stages, fifty cubic centimetres of water being permitted to enter at a time. At each stage a manometric reading is taken by turning the stopcock. These readings are plotted on a graph, in which the abscissa shows the bladder content and the ordinate indicates the intravesical pressure. The point of filling at which the first desire to void occurs is marked on the abscissa; normally this is between 150 cubic centimetres and 200 cubic centimetres. The maximum intravesical pressure obtainable by voluntary effort is marked on the ordinate; in a normal case this is 70 to 80 millimetres of mercury. Graphs are presented illustrating the types found in various pathological states of the bladder, but the author confines his deductions to the condition of the bladder wall. The claim is made that the hypertonic, normal and hypotonic bladders show unmistakable differences in their cystometrograms.

The Australian Society of Anaesthetists.

ANNUAL GENERAL MEETING.

THE first annual general meeting of the Australian Society of Anæsthetists was held at the Medical Society's Hall, Melbourne, on September 16 and 17, 1935. DR. GILBERT BROWN (Adelaide), the President, occupied the chair, and Dr. Z. Mennell (London) was the guest of honour.

Proceedings opened with the presentation of an *interim* report covering the activities of the Society since its inception in Hobart in January, 1934. The financial statement and balance sheet (published herewith) were passed, and a constitution for the Society was approved.

Dr. R. A. Hornabrook (Melbourne) was unanimously elected to honorary membership. It was decided to hold the next general meeting in Adelaide in January, 1937.

Financial Statement, 1934-1935.

	£	s.	d.
By Total Receipts	49	2	6
„ Total Expenditure	13	0	0
Credit Balance	36	2	6
Cash in Hand	5	9	8
	£41	12	2

Balance Sheet, 1934-1935.

	£	s.	d.		£	s.	d.
To Ordinary Subscriptions	20	12	6	By Stationery	1	15	0
„ Dinner Subscriptions	16	8	0	„ Bank Charges	0	5	0
„ Donations	12	2	0	„ Petty Expenses	5	10	4
				„ Credit Balance	36	2	6
				„ Cash in Hand	5	9	8
	£49	2	6		£49	2	6

(*Signed*) GEOFFREY KAYE,
Honorary Secretary-Treasurer.

Audited and found correct.
(*Signed*) GILBERT BROWN,
President.

Melbourne,
September 1, 1935.

President's Address.

DR. GILBERT BROWN read his president's address (see page 833).

Dr. Brown's paper, being a presidential address, was not thrown open for general discussion. Dr. Z. Mennell (London) was, however, asked to make some remarks.

Dr. Mennell congratulated Dr. Brown on his paper and said that only a few points required discussion. Minnitt's apparatus, even in its most portable form, was scarcely capable of use by midwives in cottage practice in, for example, the Yorkshire moorlands. Chloroform capsules had not proved satisfactory, and, if used too freely, might be dangerous. Dr. Mennell expressed a preference for gas-oxygen-ether in general surgery, as opposed to gas-oxygen alone. He described the conditions of anæsthesia in the London County Council hospitals, and regretted that up to the present the status of anæsthetists in those hospitals had not been adequately recognized. Too often they were the most junior members of the staff.

Addresses of Welcome.

DR. GILBERT TROUP (Perth) read an address of welcome on behalf of the International Anæsthesia Research Society. He presented two trophies for meritorious service to anæsthesia, bestowed by that society at its congress in Atlantic City in June, 1935. One trophy had been awarded to Dr. Gilbert Brown. The other was awarded jointly to the anæsthetic staff of the Alfred Hospital, Melbourne, and to the Director of the Baker Institute of Medical Research.

Dr. Z. Mennell delivered an address of welcome on behalf of the Association of Anæsthetists of Great Britain and Ireland. He described the organization and aims of the association, and notified its desire that the Australian Society of Anæsthetists should become affiliated with it. It was unanimously agreed that members of the society favoured affiliation, and Dr. Mennell was requested to prepare a definite scheme by which such affiliation might be arranged.

Ether.

DR. Z. MENNELL (London) read a paper entitled: "Ether is Not Dead" (see page 837).

DR. G. BROWN (Adelaide) congratulated Dr. Mennell upon his address. He pointed out that endotracheal anæsthesia for goitre cases had been almost abandoned in Australia, on account of the enhanced risk of post-operative tracheo-bronchitis. Only in exceptional cases or in emergencies was it so employed. In England, Joll alone of the surgeons whom he had seen operate, required its use. In England gas stood at one end of the scale for goitre cases; at the other end stood certain techniques which would appear strange to Australian eyes. He saw one anæsthetist, for example, proceed as follows: The patient had previously received a large dose of morphine and hyoscine. Nine drachms of paraldehyde were then administered rectally in saline solution. Anæsthesia was induced with ethyl chloride, ether and some chloroform. A large rubber endotracheal catheter was then passed and was connected up to a strange-looking assembly of glass retorts and bottles, by means of which air, ether, chloroform and oxygen were supplied to the patient.

DR. R. HORNABROOK (Melbourne) congratulated Dr. Mennell on his paper. He had used the Clover inhaler at the Melbourne Hospital and Women's Hospital thirty years ago, when its use was fashionable. A paper bag was found preferable to a rubber bag on the apparatus. Embley was largely responsible for effecting the change from "closed" ether to the "open" type of ether anæsthesia. Dr. Hornabrook preferred to use a little chloroform to assist induction with "open" ether. He regarded shielding of the eyes from light as very important, especially during induction. From experiments made upon himself he could fully appreciate the value of a comfortable posture before the induction was begun. As Sir William Willcox had remarked, every anæsthetic was a poison. Surgeons should realize this fact and should make adequately large incisions and operate with reasonable speed, so as to spare the patient as much anæsthetic as possible. Students should be taught only the simpler anæsthetic methods.

DR. D. G. RENTON (Melbourne) thanked Dr. Mennell for a very provocative paper. He considered the title, however, as rather uncalled for. Ether was, in Victoria, most certainly not dead; the majority of operations were still performed under it. He associated himself with Dr. Hornabrook's remarks. Students should be taught simple methods of ether anæsthesia; endotracheal and gas anæsthesia were definitely post-graduate work. He expressed his dislike of deep anæsthesia and of powerful mechanical retractors. Better results would follow anæsthesia as light as possible, and gentle handling of the tissues.

DR. E. C. BLACK (Adelaide) advocated the use of gas anæsthesia (nitrous oxide) in cerebral surgery, rather than ether or chloroform. He considered warmed ether vapour ineffective, in that it had returned to room temperature before it reached the patient's air passages. He favoured "Avertin" as a basal narcotic for children. He agreed with Dr. Renton that surgery should be gentle and speedy

and that ether was the anæsthetic for teaching purposes. The Clover inhaler was not used in Adelaide.

DR. F. W. GREEN (Melbourne) thanked Dr. Mennell for his paper and expressed the hope that he would shortly return to Australia, where his stimulating influence was appreciated. He asked whether the ethyl chloride-ether sequence was still employed in England.

DR. G. L. LILLIES (Melbourne) stated that in a local series of toxic goitre cases every patient for whom endotracheal anæsthesia was employed developed tracheitis and/or bronchitis. This did not apply, however, to thyreoidectomy for non-toxic goitre. For abdominal surgery light ether anæsthesia was preferable to deep anæsthesia. Endotracheal ether was the anæsthetic of choice for prostatectomy; the bleeding, if ethylene was employed, was increased. This applied, however, only to open operations on the prostate; for rapid enucleation ethylene was preferable.

DR. F. W. GREEN, speaking again, pointed out that Dr. Harris, of Sydney, had selected endotracheal ether anæsthesia as his final choice for prostatic surgery.

DR. G. BROWN (Adelaide) thanked Dr. Mennell for a useful and essentially balanced address. He himself had commenced to use barbiturates (veronal) in 1909 with very good results. On one occasion he produced unsought basal narcosis after administering thirty grains of veronal. He gave up using such large doses, not from any unfortunate personal experiences, but because of many veronal fatalities and suicides reported in the medical Press. His present routine was to give a barbiturate to each patient on the evening preceding operation. He considered that the patient should enter hospital early and be examined by the anæsthetist before 9 p.m., so that adequate rest might be secured. This preliminary visit allowed the anæsthetist to estimate the narcotic drug, its dose, and the most desirable anæsthetic (if any). Prescriptions for narcotics should be clearly written out in plain English, without medical terms or symbols, and signed by the anæsthetist. This would prevent such an error as the rectal administration of eight ounces of paraldehyde in lieu of eight drachms, owing to the nurse misreading the drachm and ounce symbols. In Australia induction of anæsthesia with chloroform was rare; induction with ethyl chloride or carbon dioxide and ether was more usual. Deaths had occurred with both the last-named, but at least the risk of primary cardiac syncope under chloroform was avoided. He agreed with Dr. Mennell that mixtures of chloroform and ether were really diluted chloroform, and had taught this since 1924. Respiratory sequelæ after ether anæsthesia seemed to be rarer in Australia than in England. Walton stated that in 90% of upper abdominal operations under ether more or less respiratory involvement might be detected. This percentage was much above that ruling in Australia. It was a good slogan to "teach ether first". In one hospital in England the students used gas anæsthesia (so-called, although actually much ether supplement was allowed) in 75% of their administrations. It would have been better to teach them sound routine practice with ether. The Clover inhaler was unpopular in South Australia, and he himself had not possessed one for sixteen years. He found open ether anæsthesia perfectly satisfactory. It had been said of Dr. Mennell that he was the only man in London who could give a good anæsthetic with the Clover apparatus.

Dr. Mennell, in reply, drew attention to veronal idiosyncrasy and mentioned that one surgeon at Saint Thomas's Hospital nearly died after taking five grains of it. The fact that chloroform-ether mixtures were really chloroform had been proved by the Royal Society in Edinburgh in 1902. Chloretone had been revived in London of late as a substitute for the barbiturates. Powerful retractors were unnecessary if the patient's muscles were lax and if the surgeon was speedy and competent. Certainly as little anæsthetic should be given as possible. He had, however, once seen Bland Sutton remove a gall-bladder under chloroform in ten minutes, and the patient recovered consciousness in five minutes more. Ethyl chloride was not much used in England now. Four deaths had occurred in one year in the casualty department of Saint Thomas's Hospital, so the drug had been discontinued there. For prostatectomy, spinal anæsthesia was rather losing ground in England.

DR. G. BROWN, speaking again, said that he had been credibly informed by ward sisters that urinary secretion was more quickly reestablished when spinal anæsthesia, rather than general anæsthesia, had been employed. Dr. Harris, of Sydney, had demonstrated prostatectomy in London, using his own technique and working with his own anæsthetist (Dr. H. Daly, of Sydney). Spinal anæsthesia was used for one patient and nitrous oxide and oxygen with ether supplement for the other.

Biochemical Aspects of Anæsthesia.

DR. IVAN MAXWELL (Melbourne), who had been specially invited to address the meeting, read a paper entitled: "Some Biochemical Aspects of Anæsthesia" (see page 841).

DR. GILBERT BROWN (Adelaide) said that he wished to thank Dr. Maxwell for his most interesting and instructive paper. It was this correlation of the scientific work of the laboratory worker with the empirical clinical observations of the anæsthetist which was so important for anæsthesia. The fall of blood pressure during an operation under anæsthesia was a matter of grave importance and could be properly determined only by frequent estimations and by making a chart of the figures. Fall in blood pressure was chiefly caused by: (i) rough handling of the tissues by the surgeon, (ii) excessive depth of the anæsthesia, (iii) spinal anæsthesia. Every endeavour should be made to prevent an undue fall, as it has been said that a blood pressure, like a woman, might be raised when falling, but not once a fall has taken place. When the circulatory depression had become large, drugs, with the possible exception of adrenaline, had little effect in raising it. This drug might be injected in doses of two minims into the muscle and followed by two minims into the skin. The action of the first injection was rapid and evanescent, but was supported by the slower action of the second. It might be necessary to use intravenous infusion of adrenaline in saline solution; but the more lasting effect of gum-saline or blood was to be preferred. Some assistance might be obtained from the use of the Trendelenburg position combined with inhalation of "Carbogen".

Both laboratory experiments and clinical work had shown the dangers of chloroform anæsthesia, and yet the fatalities were still all too numerous. Chloroform was seldom the anæsthetic of choice and might usually be replaced by a less dangerous drug. But even in the unavoidable cases it was often possible to escape primary syncope by following the method advised by Boyle more than twenty years previously. Induction should be with ether and a change made to chloroform when full anæsthesia was obtained and the risk of primary syncope had disappeared. Many years ago Dr. Brown had witnessed a death during the first two minutes of chloroform administration by a skilled anæsthetist. The appalling suddenness of the fatality and the uselessness of the methods of resuscitation remained as a vivid picture in his mind. The dangers of injecting adrenaline in a patient under chloroform were still not universally recognized, and Dr. Brown had recently been asked by a house surgeon if there was such a risk. When told of the grave possibilities, he replied: "I wondered if there was. There was a patient the other night in an attack of asthma, for which chloroform was given; he was also given some adrenaline and died straight away." Dr. Brown asked Dr. Maxwell whether this danger existed with the combination of "Avertin" and adrenaline, with which it had been said that ventricular fibrillation was produced. This was an important mater, as thyreotoxic patients were frequently given "Avertin" and then an injection was made of a local anæsthetic combined with weak adrenaline.

Dr. Maxwell had given a very illuminating account of the control of respiration. Dr. Brown wished to hear from him what was the cause of death in patients who

had inhaled an excessive amount of carbon dioxide during the induction of anæsthesia. Was it exhaustion of the respiratory centre or the effect of raised blood pressure and overload of the heart?

The introduction of insulin and the laboratory work on blood sugar had removed most of the dangers from anæsthesia in the diabetic, but it was sometimes difficult for the patient to afford the expense and hospitalization required to provide a proper blood state. In 1933 Dr. Brown had been asked to see a diabetic lady with a view to giving an anæsthetic for the removal of her tonsils. Her blood sugar was high and became still higher after her admission to hospital, on account of an attack of urticaria. It had required three weeks of careful dieting and insulin administration before her condition permitted the endotracheal use of ethylene for her operation. The dose of insulin had to be increased until she was taking seventy units three times a day. Her recovery from the operation was without any anxiety, and three months later her urine remained sugar-free on sixty units per day.

In those doubtful cases in which an immediate operation was required and there was not time for a blood sugar estimation, although the urine "slightly reduced Fehling's solution", it was well to remember that a normal person might take ten units of insulin before an anæsthetic without any danger of hypoglycæmia. This might well be followed after operation by fifty grammes of glucose buffered with twenty-five units of insulin.

Dr. Brown had been very interested in post-anæsthetic acidosis since 1909, when he saw two children die from this cause. In 1918 a patient of his suffered from severe post-anæsthetic acidosis, but recovered after intravenous infusion of glucose. In publishing an account of the case Dr. Brown had remarked the routine examination of the urine for ketone bodies had been admitted, as had also the prophylactic routine doses of glucose. A number of surgeons remarked upon this and asked if anyone really did these funny things. Since that time it had become the routine with many surgeons and anæsthetists to demand that some form of glucose should be taken freely by the patient on the day prior to operation. It might be given as syrup of glucose, which was not very palatable, or as barley sugar, orange juice or the proprietary "Glucose D".

It was interesting to find that the old order of "a pint of glucose saline" should be regarded as practically useless, as the glucose was scarcely absorbed. This was somewhat contrary to some results given by Ray, of Adelaide, who treated a series of patients, suffering from pneumonia, with rectal injections of honey in 25% solution. Some control by blood sugar estimations showed that absorption had taken place.

The investigation of renal efficiency had been instrumental in saving many lives, especially in patients suffering from prostatic obstruction. The laboratory could now inform them of the conditions on which they might make the decision whether a patient was fit for operation, whether it would be wise to continue prophylactic treatment, and also help them to choose the correct anæsthetic. The investigations of renal function would also assist in the choice and dose of the drug used for premedication.

DR. Z. MENNELL (London) congratulated Dr. Maxwell and said that the paper was the clearest and simplest exposition of the subject he had ever heard. In reference to histamine shock, Dr. Mennell drew Dr. Maxwell's attention to a case which he had embodied in his Embley Memorial Lecture, 1935. Could histadine be oxidized in the body to shock-producing histamine? Further, in deep ether anæsthesia, where the colour was of a treacherous bright pink hue, was adrenaline the best drug to give? At Saint Thomas's Hospital a mixture of adrenaline, strychnine and atropine was injected empirically in shock; this mixture would be physiologically correct if the shock was due to histamine-like substances. The combination of "Avertin" and adrenaline was commonly said to be as dangerous as that of adrenaline and chloroform. Dr. Mennell had seen no ill effects from it, although some men gave very large doses of adrenaline. He believed rectally given glucose to be useless. Intravenous infusion of 20% to 50% glucose solution had, however, a dramatic effect in shrinking the swollen, "wet" brain sometimes encountered in cerebral operations. The normal, walnut-like, convoluted appearance of the brain was restored, and the scalp might be readily closed by the surgeon over the brain thus shrunken.

DR. G. KAYE (Melbourne) likewise congratulated Dr. Maxwell. He drew attention to the value of timely intravenous infusion of saline solutions in combating surgical shock. He illustrated this by a lantern slide prepared from the operation record of a case of mesencephalic tumour. Extreme surgical shock supervened, but the fall in blood pressure was temporarily arrested by saline infusion until the more lasting effect of blood transfusion could be obtained. He asked for guidance in regard to the best form of saline infusion: whether acacia-saline, glucose-saline, or the plain isotonic saline solution advocated by McKesson.

DR. E. C. BLACK (Adelaide) asked whether a preliminary injection of ephedrine would help in obviating the shock produced by a lacerating operation, such as a radical mastectomy. He also asked whether it was possible to administer too much glucose by mouth before operation.

DR. G. TROUP (Perth) asked whether the carbon dioxide absorption method of anæsthesia might deleteriously affect the pH of the blood.

Dr. Maxwell, in reply, said that he was particularly interested in allergic phenomena. He thought that Dr. Brown used too small doses of adrenaline, and suggested that in asthma two minims might be given every minute for even fifty minutes if necessary. Intravenous administration of adrenaline was, he considered, dangerous. Hence if an asthmatic were giving himself injections of adrenaline, he should be taught to test by aspiration that the needle was not in a vein. Even normal people might show cardiac irregularity after injections of adrenaline, and intravenous administration might well precipitate ventricular fibrillation. He had had no experience of the "Avertin"-adrenaline combination. Excessive administration of carbon dioxide might elevate the blood pressure, which might possibly lead to dangerous dilatation of the right side of the heart.

In reply to Dr. Mennell, he said that histadine was converted to histamine in the body. He thought that strychnine was of little value in therapeutics. In regard to intravenous saline therapy, the rise in blood pressure so produced in laboratory animals was marked, but evanescent. Hence, on physiological grounds, saline infusion should be followed by blood transfusion. Even so, some curious ill-effects had followed the transfusion of blood, even after careful typing of donor and recipient. This subject was, however, too big to discuss. The carbon dioxide absorption technique would have some slight influence on the pH of the blood, because the respiratory centre would react sensitively to the carbon dioxide withdrawal. Only very gross abnormalities, however, would make any material difference in the reaction of the blood. The oral administration of glucose, sometimes for two or three days prior to operation, and perhaps in combination with insulin, was now a recognized technique with modern anæsthetists. Ephedrine, when injected, often caused tremor and apprehension; hence, if given pre-operatively, as suggested by Dr. Black, it should be combined with a barbiturate or other sedative.

Sources and Pharmacology of Impurities in Anæsthetics.

DR. B. L. STANTON (Melbourne) read a paper entitled: "Sources and Pharmacology of Impurities in Anæsthetics" (see page 845).

The Use of Carbon Dioxide in Anæsthesia.

DR. E. C. BLACK (Adelaide) read a paper on the use of carbon dioxide in anæsthesia (see page 849).

DR. D. G. RENTON (Melbourne) thanked Dr. Black for his paper in which many problems of interest were very ably reviewed.

Apart from two references to physiology, Dr. Renton proposed to base his remarks on his own experience in the evolution of the technique and on the ideas that he had adopted in regard to this gas and its relation to anæsthesia. In some phases the ideas of Dr. Black and himself were closely similar, but in others there was some considerable difference of opinion; but as this was to be a discussion, he hoped to stimulate others to expound their views.

Dealing first with physiology, Dr. Renton said that in the ordinary course of events the mechanism for the regulation of respiration was adjusted to function in the presence of some 0·04% of carbon dioxide in the atmosphere. The raising of the content of carbon dioxide to 0·1% caused that atmosphere to become stuffy and oppressive, this figure being the criterion on which ventilation requirements were based (Starling, "Principles of Physiology").

In the second place the addition of quite small proportions of carbon dioxide to the atmosphere produced definite increase in lung ventilation. The following figures were quoted from Starling. If it were assumed that the lung ventilation in the presence of the normal atmospheric 0·04% carbon dioxide was 100 (6·6 litres per minute), raising of the carbon dioxide to 0·79% increased lung ventilation to 116. Carbon dioxide 2·02% increased it to 153; 5·14% increased it to 498, and 6·02% to 857. It appeared that the alveolar air content of carbon dioxide was not increased until there was over 5% carbon dioxide in the atmosphere. From these two points two conclusions might be drawn: (i) that the addition of a very small percentage of carbon dioxide to the inspired atmosphere produced increased lung ventilation; (ii) that quite a small increase in the carbon dioxide content of that atmosphere rendered it unpleasant.

Dr. Renton had commenced using carbon dioxide with ether anæsthesia late in 1928. The first feeling was that if one stimulated the respiration, thereby increasing lung ventilation, the time of induction would be shortened. It was found that this was so. But from the patient's point of view was there any advantage? Until consciousness was lost there was a distressing feeling of shortness of breath, tremendous pounding of the heart—altogether rather a stormy period. Furthermore, there was undoubtedly an increased secretion of saliva and mucus. These effects more than counterbalanced the saving in time. Time was surely of secondary importance, the comfort of the patient being the anæsthetist's main concern. Certain advantages of carbon dioxide over ethyl chloride had made him reluctant to give it up completely, so he had adopted a modification in its use.

First, the simple apparatus shown was devised. It consisted of a small rubber bag which was filled from the carbon dioxide cylinder. From it a tube passed under the mask, with a regulating tap between mask and bag. When distended, this bag held perhaps three litres of pure carbon dioxide. The mask, draped with towels and sprinkled with a few drops of a mild scent, was placed in position and the patient was instructed to relax as for sleep and to breathe quietly. A slow stream of carbon dioxide was then turned on. The rate of flow was visible by the rate of deflation of the bag. After a few breaths ether was added in single drops. The rate of dropping was slowly and progressively increased until the mask was sufficiently saturated.

The flow of carbon dioxide was maintained throughout the ten minutes or so taken by the induction, in the average case less than two litres being used; hence the actual addition of carbon dioxide to the inspired air was much less than 1%. This amount caused no violent respiratory stimulation and produced no unpleasantness for the patient. It did, however, regularize the respiration, which was slightly deepened, but not quickened. It was sufficient to concentrate the respiratory centre on respiration, the tendency to gagging and spluttering and irregular respiration was restrained, but without unpleasantness.

This gentle persuasion of the respiratory centre with minimal amounts of carbon dioxide *plus* the slow addition of ether rendered induction as quiet and pleasant as any ether induction could be. The majority of patients quietly fell to sleep and then passed into anæsthesia. This did not imply over-sedation prior to anæsthesia. Most of the patients had one-sixth of a grain of morphine with one one-hundred-and-fiftieth of a grain of atropine an hour beforehand, with an overnight dose of barbitone, producing merely mild sedation, not basal narcosis.

The pleasantness of this technique was evident by the large proportion of patients who so quietly went to sleep, and by the rarity of struggling; it was favourably commented on by patients, including *confrères*, who had had previous inductions with ethyl-chloride-ether sequence or ether alone. One patient had described it as marvellous; she was to be forgiven, because she had had a previous induction with the Clover inhaler and was terrified.

As soon as anæsthesia was established, the flow of carbon dioxide, which, by the way, had been becoming less and less with deflation of the bag, was completely shut off; no more was given unless conditions arose in which it was needed. These conditions often resulted from a too deep plane of anæsthesia being reached, and, if this were so, indicated a fault in technique.

Dr. Renton did not approve of any meddlesome use of carbon dioxide without very definite indications. Any prolonged stimulation of the respiratory centre, no matter of how mild a degree, necessarily produced a corresponding degree of fatigue of that centre. Their purpose was to save the patient the expenditure of any unnecessary energy, and unnecessary stimulation by carbon dioxide did cause a needless wastage. With the perhalation method an excessive accumulation of carbon dioxide was never encountered, and the problem of getting rid of it did not arise as it did in closed system methods of anæsthesia. Even so, an occasional breath of pure air was advantageous.

To revert to the Clover inhaler. Induction of anæsthesia by this technique was certainly rapid. But what a horror to the patient! There was a rapidly increasing stimulation by accumulating carbon dioxide, a rapidly decreasing supply of oxygen, with a superimposed addition of pungent ether vapour; the result was a terrifying experience for the patient.

Dr. Black had mentioned the use of 30% carbon dioxide in oxygen by others as a means of inducing anæsthesia. Dr. Renton's only comment was: "barbarous".

Carbon dioxide problems arose in connexion with endotracheal or endopharyngeal methods of maintenance of anæsthesia.

Referring to endotracheal insufflation, Dr. Renton said that in this technique, when efficiently carried out, the patient was supplied with an amount of air considerably in excess of the respiratory requirements, to which was added an adequate amount of anæsthetic vapour. The over-supply insured a constant outflow of air through the larynx, even during the phase of inspiration, thereby partially protecting the bronchial tree from the entrance of foreign matter. As the supply was delivered into the trachea itself, there was no dead space for any rebreathing to occur, to retain carbon dioxide, and this of necessity caused a lowering of the alveolar tension of carbon dioxide with consequent tendency to alkalæmia. The muscular effort required to expire through the narrowed exit and against the pressure of the insufflated air caused increased oxygen consumption and increased excretion of carbon dioxide, which partially counteracted the constant washing out of alveolar carbon dioxide by this over-ventilation. Should signs develop indicating depletion of carbon dioxide, a very small quantity might with advantage be added. Dr. Renton's practice was to mix with the air a small stream of 5% carbon dioxide in oxygen. This small amount compensated the excessive washing out of carbon dioxide.

At the termination of the anæsthesia, with return of reflexes and withdrawal of the catheter, there was often a period of respiratory sluggishness caused by the alkalæmia produced; and a degree of anoxæmia, possibly with cyanosis, might develop. The judicious use of carbon dioxide in minimal proportions would tide a patient through this period. If any excessive respiratory excursus was produced in aiding deetherization, it was well for the

anæsthetist to guide the respiration carefully back to normal before allowing the patient to pass out of his care. To a lesser degree these remarks applied to any method of endopharyngeal anæsthesia.

In regard to endotracheal inhalation, success of the technique required the use of the widest bore catheter which the larynx would accommodate, and the establishment of a closed system by pharyngeal packing, if necessary, between the respiratory system and the anæsthetic apparatus, insuring absolute protection of the bronchial tree from the invasion of foreign material from the pharynx. The expiratory valve and volume of supply had to be adjusted so that there was adequate escape of the products of expiration and sufficient supply of air to insure ample removal of carbon dioxide and maintain efficient oxygenation, otherwise a sufficiency of carbon dioxide to produce respiratory stimulation, with consequent waste of muscular effort, sweating and loss of body heat, would soon accumulate. On account of the adequate airway provided by the large catheter and the low pressure in the system, respiration was carried on with the minimum of effort by the patient. Relaxation was good with relatively light planes of anæsthesia, oxygenation was perfect, and there was less loss of energy, fluid or body heat than by the insufflation method. Nor was there depletion of carbon dioxide with alkalæmia.

In regard to gaseous anæsthetics, except in cases of nasal administration and the rare occasions on which the endotracheal insufflation technique was used, they were administered from a closed system, with provision for exit from the system through an expiratory valve.

If they imagined a patient breathing at the rate of twenty respirations per minute, of tidal capacity of 500 cubic centimetres, into a closed system of ten litres capacity, containing at the outset some 20% oxygen and no carbon dioxide, it would be found that carbon dioxide was built up in the system at the rate of approximately 5% per minute, and the oxygen was depleted at approximately the same rate. To obviate the building up of carbon dioxide within the system there were three available procedures.

1. Complete emptying of the system at frequent intervals (each two minutes or so) and refilling with fresh gas mixture. This necessitated the wastage of an enormous amount of gas, and there was a constant variation in the gas mixture in use.

2. Partial rebreathing, whereby portion of each expiration was allowed to escape and be replaced by fresh gas mixture. Gas wastage was fairly extensive in this method, but less so than with the first. The late Dr. McKesson had advised rebreathing up to within 200 cubic centimetres of the tidal capacity of the lung, which was determined from time to time throughout the anæsthesia. With all due deference to McKesson, Dr. Renton considered this to be over-rebreathing. Dr. Renton did not allow the patient to rebreathe more than one-fifth of the tidal capacity when using McKesson's apparatus.

3. The "closed-circle" technique with absorption of carbon dioxide by means of soda lime. In this method the expiration was passed through soda lime, which extracted most of its carbon dioxide. Oxygen was added in a continuous flow of sufficient volume to maintain adequate oxygenation. After induction the only gas added was that necessary to replace any leakage from the apparatus. From the economic aspect it considerably reduced the actual costs of anæsthetic material used.

In the absence of stimulation by carbon dioxide, respiration was quiet and, provided a free airway was obtained, muscular relaxation was adequate for abdominal surgery with quite light anæsthesia. Oxygenation was excellent, the blood being kept bright throughout and no anoxæmia being present. There was practically no mucous secretion. There was no respiratory effort, no wastage of energy, no sweating, no loss of body heat from the anæsthetic *per se*, and there was frequently no post-anæsthetic vomiting. Most patients recovering from gas anæsthesia vomited or retched at least once with the return of consciousness, although memory of this was very often not retained.

Dr. Black had alluded to shock and the various theories of its causation. They were all familiar with the clinical picture of operative and post-operative shock (some called it anæsthetic and post-anæsthetic shock)—the cold, moist skin and general depression of all vital functions. The multiplicity of theories propounded as to the causation of this state indicated that knowledge of its ætiology was meagre.

In fact, Dr. Renton did not see how any one single factor could account for the condition. It was surely the cumulative result of many factors, all acting in the same direction, causing depression of all vital functions. They were all probably agreed that a feature of shock was the withdrawal of fluid from the circulating blood stream.

So far as the anæsthetist himself was concerned, there were three factors well within his control by which he might lessen the tendency to shock. The first was the use of as little anæsthetic as possible. The second was by the provision of free ingress to and egress from the lungs of the anæsthetic mixture. Dr. Renton did not allude to gross degrees of obstruction. There were minor degrees of obstruction which produced an increase in resistance to the expiration. Any resistance at all meant that the patient used extra muscular effort to carry on respiration. This required an increased oxygen consumption and increased carbon dioxide output. This increased use of energy was accompanied by sweating, whereby body fluid was lost, and by evaporation of sweat body heat was also lost. With expiratory resistance alone there was just this wastage, but with inspiratory resistance there was added a severe strain on the myocardium by virtue of the vacuum action of the thorax, by which, in the presence of inspiratory resistance, there would be a tendency to over-filling of the heart. The cumulative effect of this over any length of time must be harmful and must act as a considerable factor in producing the shocked condition.

The third factor within the control of the anæsthetist was that stimulation by carbon dioxide, even if only slight, imposed the necessity for added muscular effort, with consequent waste of energy, fluid and heat. Dr. Renton's aim was to prevent stimulation by carbon dioxide, unless conditions arose in which it might be essential to tide over some temporary emergency.

In the sudden emergencies which the anæsthetist occasionally met, when, to save life, active and rapid stimulation was necessary, carbon dioxide, provided it could reach the blood stream and in the presence of a sufficiency of oxygen, was the best and surest stimulant available. Its primary effect was on respiration, and with any improvement of respiration there must be, *ipso facto*, an improvement in the cardio-vascular function. In crises of this type it was well to avoid over-stimulation, either by carbon dioxide or by any other drug, such as adrenaline, ephedrin, "Coramine", "Alphalobeline". A certain amount had to be left to Nature. It was easy to fall into the trap similar to that of the driver of the tired horse, who, by vigorous application of the whip, finished off his horse.

The use of carbon dioxide in the post-operative period was extremely useful. The establishment of full expansion of the lung at the termination of anæsthesia would open up any atelectatic patches and thereby lessen the likelihood of post-operative chest complications. It was well to repeat this full expansion at intervals throughout the first twenty-four hours, or longer if necessary. This could be well accomplished by a 5% carbon dioxide mixture with oxygen.

To Dr. Renton it seemed that concentrations of carbon dioxide greater than 5% were practically never necessary. The use of stronger concentrations and of pure carbon dioxide should be undertaken solely by the anæsthetist himself. A concentration of 5% of carbon dioxide in oxygen was reasonably safe and was suitable for use by nurses after instruction and demonstration of the effect it was desired to produce.

Dr. Renton did not approve of prolonged stimulation of respiration, and once full expansion had been established the carbon dioxide should be withdrawn and respiration

guided back to normal. Here again a good deal must be left to Nature.

If they provided an adequate supply of fluid, kept the patient warm and as comfortable as possible, Nature would accomplish much. Meddlesome therapeusis frequently did more harm than good.

DR. G. BROWN (Adelaide) thanked Dr. Black for the enormous amount of work which he had evidently put into his paper. He recalled that the pioneer work on carbon dioxide was actually done by Hickman in the pre-anæsthetic era. He (Dr. Brown) had early used the gas for speeding up the induction of anæsthesia, and had learned from his consequent mistakes. He did not now administer it as a routine, although at times it was extremely useful. When using the Heidbrink apparatus for dental gas anæsthesia, however, he preferred to attach a cylinder of carbon dioxide and oxygen mixture rather than a cylinder of oxygen. It had been said by Lidwill, of Sydney, that deetherization with carbon dioxide raised instead of lowered the incidence of post-operative respiratory sequelæ. Used in reason, however, Dr. Brown found it very valuable. He instructed house surgeons to employ the carbon dioxide and oxygen mixture for three minutes every three hours, for thirty-six hours after operation, in cases in which anxiety was felt for the lungs. Autopsies performed in the Adelaide Hospital upon patients dying of post-operative respiratory sequelæ revealed a high incidence of undiagnosed massive collapse of the lungs; deetherization therefore was a valuable routine procedure.

Dr. Black, in reply, said that he had not yet encountered any case of massive collapse of the lungs. When he administered carbon dioxide he liked to employ a sight-feed flowmeter. He disagreed with Dr. Renton as to the universal desirability of light anæsthesia; many surgeons were of opinion that deep anæsthesia, at certain stages of the operation, was more effective in mitigating shock. Carbon dioxide gas was too potent to be entrusted to nurses; in such event the carbon dioxide and oxygen mixture was safer.

Anæsthesia in America.

DR. G. TROUP (Perth) read a paper entitled: "Anæsthesia in America" (see page 857).

DR. G. BROWN (Adelaide) said that all members must feel much indebted to Dr. Troup. None were, however, capable of discussing cyclopropane. He (Dr. Brown) had seen it employed in England. There some fear was felt that it might damage the liver, and a limit of one and a half hours had been proposed for its administration. In this respect it was possible that the English workers were not acquainted with current researches made in America. He had been informed in England that, should the gas obtain general vogue, it could be produced there much more cheaply than in America.

Dr. Troup then demonstrated the latest model of the Waters-Foregger "midget" gas anæsthetic apparatus, intended for the administration of cyclopropane or ethylene by the carbon dioxide absorption method. He also demonstrated Paul Wood's apparatus for the administration of "Avertin", to which he had referred in his paper.

Choice of Anæsthesia in Some Surgical Conditions.

DR. G. L. LILLIES read a paper entitled: "Choice of Anæsthesia in Some Surgical Conditions" (see page 863).

DR. G. KAYE (Melbourne) congratulated Dr. Lillies upon a paper which, as might be expected of him, was definitely provocative. Dr. Kaye believed that in minor dental cases patients might well receive gas anæsthesia by the nasal route, but in major cases the endotracheal method with ether or gas anæsthesia was called for. A "minor" case was, however, hard to define. He himself would put it as one occupying not more than ten minutes, or as involving the extraction of six or eight teeth likely to involve no technical difficulties and no great hæmorrhage. In cases other than the above the patient should receive endotracheal anæsthesia for protection of the larynx, and should be operated upon in hospital. Tonsillectomy under endotracheal gas anæsthesia was not very agreeable for the surgeon, as the cough reflex was hard to control and the large catheter was an annoyance. Dr. Kaye was, however, obliged to handle a fairly large number of such cases in the Alfred Hospital, mainly in patients requiring tonsillectomy for acute nephritis. He agreed with Dr. Lillies that bleeding was probably greater under gas than ether, and that the blood (though not cyanotic) seldom had the redness typical of the blood under ether. This might, in sinus operations, obscure the rays of the frontal lamp and embarrass the surgeon. In "open" nose and throat operations, however, such as mastoidectomy, gas anæsthesia was perfectly satisfactory; in the last-named operation it need not necessarily be given endotracheally. There was therefore no reason to use a drug so toxic as chloroform for such patients. In cerebral surgery the patient often "hung by a hair", and gas anæsthesia was thus desirable, owing to its lack of toxicity. In Dr. Hugh Trumble's department at the Alfred Hospital, about 35 cerebral operations had been performed upon 28 persons in the last eight months alone. Local anæsthesia had initially been favoured, but premedication had proved a difficulty. If the patient were fully premedicated, his circulation and respiration were apt to be depressed. If inadequately premedicated, he had to face a terrible psychological strain. Dr. Trumble was asking, therefore, for general anæsthesia in an ever greater percentage of patients, possibly in 50%. This was especially the case in patients with raised cerebral tension, particularly those having lesions in the posterior cerebral fossa. Respiration was here liable to great depression, and the presence of an endotracheal tube was so helpful that general anæsthesia was adopted in order to secure it. The increased bleeding said to occur in cerebral operations under gas seemed to Dr. Kaye to be mythical. Once the skin had been incised and the bone flap raised, concentrations of oxygen approaching to 85% could be given in cerebral cases. Dr. Kaye produced in illustration "Nargraf" records of two patients: (*a*) one undergoing removal of the Gasserian ganglion, in whom the average oxygen requirement was 20%; (*b*) one undergoing cerebral exploration, in whom the average oxygen requirement was 85% once the brain had been exposed. In thoracoplasty surgical shock was great, owing to extensive laceration of tissue. Local anæsthesia here added a large psychical strain. Dr. Trumble had at one time been very interested in this operation, and the technique finally evolved included: (i) posturing the patient so that he rested upon his diseased lung, (ii) gas anæsthesia at positive pressure, but not necessarily by the endotracheal route, (iii) limitation of the operation to short stages, three or four ribs being removed at each stage. Dr. Kaye had had no experience of lobectomy, and his experience of anæsthesia for thoracic surgery was limited to endotracheal gas anæsthesia at positive pressure. He did not propose to reopen the perennial argument as to the relative advantages of nitrous oxide and ethylene in abdominal surgery. With water-flowmeter machines he preferred ethylene. With dry-flow, positive-pressure apparatus either gas seemed to him to give equally good results. He had a slight personal bias in favour of nitrous oxide.

DR. A. D. LAMPHEE (Adelaide) thanked Dr. Lillies for his paper and was pleased to hear him praise ether. This was, and would for long remain, the routine anæsthetic in Australia. He found nitrous oxide difficult for goitre cases, and preferred ethylene. He asked Dr. Lillies what he would advise for a fat patient with intestinal obstruction and fæculent vomiting. Local anæsthesia might not insure sufficient relaxation, and the presence of vomiting would render gas anæsthesia dangerous. Would spinal anæsthesia be best?

DR. D. G. RENTON (Melbourne) congratulated Dr. Lillies on his wide experience of thyreoid surgery, and agreed with him in all he said of it. An endotracheal catheter should, however, always be at hand in such cases for use in emergency. In one week recently Dr. Renton had encountered three such emergencies: (*a*) A case in which, entering the theatre, he found one of his fellow anæsthetists in difficulties. The patient's respiration and

circulation had become suspended following collapse of the trachea; fortunately, animation was restored after intubation of the larynx and insufflation of oxygen. (*b*) On the next day he encountered a similar case, requiring similar treatment. (*c*) On the day immediately afterwards he met with a third case presenting similar symptoms and was saved from the necessity for laryngeal intubation only by the administration of the anæsthetic under pressure from a McKesson machine. Dr. Renton liked the McKesson apparatus for thyreoid, cerebral and thoracic surgery, but for general surgery preferred the water-flowmeter apparatus, since he considered that (with the former) the patient had to exhale against a gaseous pressure and thereby exhausted much energy. He regarded toxic goitre patients as the worst anæsthetic "risks", because they presented an unknown factor which could not be determined by laboratory tests. Patients for prostatectomy caused nearly as much anxiety, and could be assessed only by the anæsthetist's "sixth sense" of impending trouble. After seeing many surgeons use the Harris operation, he considered that it involved a longer operation time and therefore more operative shock than enucleation involved. He preferred ethylene as the anæsthetic, given by mask or (if the respiration were at all embarrassed) endotracheally. He believed that 25% of his prostatic patients would have succumbed under any other anæsthetic.

Dr. Lillies, in reply, agreed with Dr. Hotten that surgeons should consult the anæsthetist more than they did regarding the choice of anæsthetic and technique. They were, however, becoming more cooperative in this and in allowing the anæsthetist to visit the patient before operation. He had never seen retrosternal pressure symptoms in cases of toxic goitre, although he had often met with them in non-toxic goitres. He had seen one such patient succumb, apparently from acute distension of the right side of the heart, despite intubation of the larynx. He disagreed with Dr. Kaye that six or eight teeth might be removed in a dental surgery; he would set a limit of three teeth. He considered, too, that with proper psychological handling (at least as important as proper premedication) patients could be rendered cooperative in respect of cerebral and thoracic operations under local anæsthesia. In answer to Dr. Lamphee, he had seen good results from local anæsthesia in cases of intestinal obstruction, but would regard spinal anæsthesia as the technique of choice. He agreed with Dr. Renton that an endotracheal catheter should be at hand at every goitre operation; indeed, at every operation. In goitre cases especially one could not tell, without prior laryngoscopy, whether one recurernt laryngeal nerve might not be paralysed. Should the other be divided by the surgeon, dangerous dyspnœa would result and would call for prompt intubation. He had not actually seen tracheal collapse, although he had heard of it. He suggested that the symptoms attributed to it might be really due to paralysis of the vocal cords. For thyreoid surgery he still employed the earliest type of "Austox" apparatus; it had no flowmeters, and gas was added directly from the cylinders as required.

DR. D. G. RENTON, speaking again, said that in the cases of "tracheal collapse" encountered by him there was no evidence of vocal cord lesion. The cords on laryngoscopy were widely apart and the respiratory obstruction appeared to occur at a lower level than the glottis.

Chloroform.

DR. J. BEAN read a paper on chloroform. He dedicated it to the memory of his old teacher, Richard Gill, chloroformist to Saint Bartholomew's Hospital from 1881 to 1913. He described Gill as a man of emphatic personality, staccato in manner and incoherent in expression, as though tripped up by his own rapid thoughts. He was by some regarded as the best anæsthetist of his day in London, and as perhaps the most expert chloroformist on record. His use of rebreathing with the Clover inhaler had antedated the ideas of Yandell Henderson. In 1906 Gill published two volumes, embodying his life's experience, which he entitled "The $CHCl_3$ Problem". This work was superficially eccentric, full of oddities of style and expression, of metaphysical speculations, of quasi-mathematical formulæ, which tended to bewilder and repel the casual reader. It had passed, accordingly, almost unnoticed when published, and was now practically forgotten. Dr. Bean, however, regarded it as the most valuable of all single books on anæsthesia. He pointed out that it embodied the result of very wide clinical experience. Gill did not favour argument by analogy from animal experiments. He conducted his observations on the human subject, tabulating and analysing observed clinical phenomena. From these observations he produced certain logical conclusions which Dr. Bean presented in his paper. The conclusions in turn were checked by *in vitro* experiments upon human blood, it being Gill's belief that the physiological action of chloroform was one of "deoxygenation" and that this action was exerted upon the red blood corpuscles.

Dr. Bean pleaded for a study of Gill's book as a preliminary to any further research on anæsthesia in general, and on chloroform in particular. He pointed out that a revived interest in chloroform was desirable, since the possibility of war was imminent, and chloroform, being portable and convenient, had a large place in military anæsthesia.

Dr. Bean proceeded to outline Gill's argument. In physiology every active agent produced a characteristic syndrome of effects, varying according to the intensity of action of the exciting agent. If chloroform was regarded as a physiological agent, two resulting syndromes could be traced: (*a*) the routine characteristic effects; (*b*) certain crises, such as arrest of circulation or respiration, or convulsions in the induction period. Gill, noting that such crises were only occasional and had no definite time sequence in the periods of induction and maintenance, had come to regard them as something apart from the true physiological action of chloroform. What, then, were they? Were they physiological effects quite apart from the chloroform syndrome, or were they chloroform effects modified by extraneous factors, thus producing the crises? If the latter, it would be necessary to identify and eliminate the extraneous modifying factors before the true action of chloroform could be appreciated. It was to discover this true physiological action and the part of the body acted upon that Gill set himself.

Gill had first distinguisbed between anæsthesia (the lightest degree of surgical unconsciousness, and characterized by automatic respiration) and certain deeper grades of narcosis. He then proceeded to standardize in his own mind the exact clinical phenomena found in the induction and maintenance of standard normal anæsthesia and the various planes of narcosis. Phenomena encountered which did not fit into these categories were, he thought, caused by something else than pure chloroform action. He had next to tabulate those factors which, with and without chloroform's complicity, were present in the "crises" above mentioned.

Gill found that anæsthesia, as he defined it, was always perfectly safe in reasonably skilful hands, apart from the rare chance of primary cardiac syncope. Narcosis, on the other hand, was always potentially dangerous, especially if prolonged. This danger lay, not in any direct toxic action of chloroform on heart muscle, heart innervation or respiratory centre, but in a mal-aerated and stagnating alveolar circulation. This produced a vicious circle of anoxia, which exhausted the cardiac and respiratory musculature.

Primary chloroform deaths, Gill held, were rare, and were always due to anoxia. This was borne out by the rapid circulatory failure of "back-telling" type, which was seen clinically in such cases, and by the engorged condition of the right side of the heart and venous system found at autopsy. Further, if resuscitation were successful, the return to normal was too rapid for the syndrome to have been due to direct intoxication of the cardiac muscle or innervation; such intoxication would need longer time for recession. Chloroform crises were always "mixed" crises—a complex of chloroform *plus* certain

intercurrent critical factors to be later described. Such factors were much more likely to act in narcosis than in true anæsthesia.

The physiological action of chloroform was to rob the red corpuscles of nascent oxygen, the drug being split thereby into inert decomposites. Anoxæmia resulted, with impaired tissue oxygenation. Brain tissue, delicate and richly vascular, and striped muscle were especially sensitive to oxygen-lack, and responded to it by diminished function, thus producing anæsthesia. The anoxæmia produced by chloroform did not, in anæsthesia, affect the normal colour of the blood, although cyanosis was progressively present in narcosis. Gill found that shed blood exposed to graded concentrations of chloroform vapour *in vitro* became redder than normal, because the corpuscles resisted the deoxygenating action of the chloroform by clumping into refractile and reflecting rouleaux. Higher concentrations of chloroform, as in narcosis, broke up the rouleaux, made the corpuscles shrink, reduced much of the oxyhæmoglobin, and gave to the blood a cyanotic, mahogany colour. Shaking the cyanotic and venous blood with air restored its red colour. Gill concluded, then, that chloroform abstracted oxygen from the blood and was itself oxidized in the process, since a drachm of chloroform shaken with a pint of blood lost all its characteristic odour. Further, the phenomena of chloroform crises were all (save discoloration of the blood) due, not to the chloroform, but to the associated anoxæmia.

In anæsthesia the lung alveoli contained less than their normal quota of air, which was partly replaced by vaporized chloroform. This anoxæmic tendency was counteracted by increased rate of respiration. In narcosis, however, respiration slowed and a "negating" process was set up, tending to promote anoxæmia of the alveolar blood, hence of the whole system. This anoxæmic tendency had to be distinguished from the physiological deoxygenating action of chloroform. The latter was associated with a sluggish, slowly dilating pupil. The alveolar oxygen negation, however, was associated with an active pupil, which, when it did dilate, dilated suddenly and to the full. Again, in pure chloroform anæsthesia respiration was automatic because the cerebral centres were released from higher control. In alveolar obstruction, however, the respiration was laboured and never automatic.

Chloroform was four and a half times as heavy as air. Hence it tended to form a high-concentration pool in the lower bronchial tree. Should mechanical respiratory obstruction occur, this pool readily produced "negation" anoxæmia and overloaded the blood with chloroform, intensifying the normal deoxygenating action of the drug. Further, the weight of the pool might of itself embarrass respiration, producing progressively laboured respiration of a more and more diaphragmatic type. Restoring the patency of the airways and sharply squeezing the thorax would restore normal thoracic respiration.

Respiratory arrest might be due to: (*a*) absolute excess of chloroform, acting both physiologically and by negation effects, and by its mechanical presence, and (*b*) relative excess, due to respiratory subnormality, as in the shallow respiration of fear or impending vomiting. It followed, then, that a given crisis might be due to many factors—to the dose of the drug itself, to mechanical respiratory obstruction, to a muscular mechanism debilitated by cachexia or fatty degeneration, and to vagaries in depth of respiration. Such factors were worthy of detailed analysis.

Mechanical obstruction had many obvious causes. To them should be added muscle atony due to fainting and severe hæmorrhage. Gill had curiously, Dr. Bean pointed out, overlooked the influence of the glairy mucus secreted by the air passages as a protective against the tri-chlor radicle of chloroform, and which might produce insidious respiratory embarrassment. It might be mitigated by the use of atropine premedication, by gradual induction and by a gauze wick oral drain. Gill had never recognized the value of atropine as a precaution against vagal inhibition. Embley, who first isolated this cause of chloroform crises, wrote some years after Gill.

Emotionalism favoured choloform overdose, because it led to shallow breathing. A few breaths of ether might stimulate effective breathing. Emotionalism had even caused death from pure terror (possibly by vagal inhibition) before any chloroform had been given.

Stomachic inhibition of respiration occurred when the stomach was handled or as a prelude to vomiting. Respiration was shallow, with secondary vasomotor pallor, and the shallow breathing might induce chloroform overdose. Especially would it do so if mechanical respiratory obstruction or alveolar congestion existed and was not quickly relieved. Cessation of stomach traction might afford relief. The chloroform dosage should be cut down to match the shallow respiration, and, if anæsthesia required to be deepened, ether should be temporarily used.

Reflex respiratory arrest was most usually met as a prelude to vomiting or in emergency surgical patients with a full stomach. It occurred also in intestinal obstruction, in handling of the stomach and when much blood or mucus had been swallowed. Characteristically, respiration failed at the end of a full inspiration. The diaphragm became fixed, the stomach emptied itself, and respiration was soon resumed. This occurred, however, only in true and perfect anæsthesia. In narcosis respiration might fail at any stage of inspiration or expiration and could not recommence without help, because of the congested pulmonary circulation. Artificial respiration should therefore be commenced; when it had overcome the anoxæmia, a full and spontaneous respiration would occur, followed by vomiting and by normal respiration. The fact that the respiratory centre so soon resumed activity showed that it was embarrassed by anoxæmia only, and not directly poisoned by chloroform; in that event it would have been crippled for a far longer period.

Reflex stimulation of the vasomotor centre, according to Gill, was the general cause of circulatory crises. The main part of the systemic blood was at once shunted into the splanchnic pool. In itself this was merely a reflex "faint" and was innocent if anæsthesia (rather than narcosis) were present, if the airways were patent and if no "pool" of chloroform formed therein. When the splanchnic pool filled, the heart, having little systemic blood to circulate, reduced its activity; the muscles, being exsanguinated, were wholly relaxed. It was necessary, therefore, first to insert an artificial airway to counteract obstruction due to relaxed muscles. The patient should thus be up-ended to cause gravitation of blood from splanchnic vessels to the *vena cava* and so back to the heart. Cardiac stimulants were unnecessary. The pulse would soon return and the circulation reestablish itself, proving that the condition was not due to direct cardiac intoxication by chloroform. If narcosis, however, were present when the crisis occurred, anoxæmia also would be present, and this, especially if combined with a glairy mucous plug, might have a fatal issue.

Gill cited also a "law of diminishing resistances". By this, a dose of chloroform, anæsthetic in the earlier stages of administration, grew narcotic by the later stages. Gill held that the blood corpuscles directly wilted in their resistance to the deoxygenating action of the chloroform, whilst other tissues did so in consequence of nutritional disturbance due to deoxygenation during a long administration. This law, according to Gill, explained many fallacies in describing the action of chloroform. Such fallacies arose through confusion of the constant chloroform effect with the variable chloroform result—the product of the chloroform effect with the status of the parts affected, which status varied according to the law of diminishing resistances.

Gill concluded, then, that the blood was the prime seat of the physiological action of chloroform, and that the remoter effects of the drug were secondary and anoxæmic. Dosage therefore should depend on the quality and quantity of blood in circulation at any given time. Rapidity of induction, however, should be in proportion to the efficiency of the respiratory mechanism, provided that the optimal dose was not exceeded. That dose, in the last analysis, would depend upon the amount of oxygen that it was requisite to be extracted from the blood.

Dr. Bean concluded by remarking that Gill had shown chloroform to be anything but foolproof. It, more than any other general anæsthetic, depended for its safety on the man using it. Men unprepared to acquire the necessary knowledge and technical mastery would be well advised to leave chloroform alone. That its administration could be mastered was proved by the careers of the elder Symes, of Lister and of Gill, all of whom professed to have used the drug many thousands of times without fatality. In conclusion, Dr. Bean urged his audience to explore for themselves the originalities, the practically useful and theoretically interesting "clinicalia", the shrewd inferences to be found in Richard Gill's "The $CHCl_3$ Problem".

Anæsthetic Deaths.

DR. GEOFFREY KAYE read a paper in which he discussed the anæsthetic deaths that had occurred in Melbourne during the period 1929-1934.

A member who wishes to remain anonymous read a paper on anæsthetic fatalities (see page 866).

British Medical Association News.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Turnbull, Harley Irwin, M.B., B.S., 1929, B.Sc., 1928 (Univ. Sydney), F.R.C.S. (England), 1934, Pritchard Street, Wentworthville.

Obituary.

JOSEPH ERNEST GOOD.

WE regret to announce the death of Dr. Joseph Ernest Good, which occurred at Adelaide, South Australia, on December 6, 1935.

NIGEL ALLAN ALLISON TRENOW.

WE regret to announce the death of Dr. Nigel Allan Allison Trenow, which occurred at Townsville, Queensland, on November 27, 1935.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii, xix. and xx.

MOOROOPNA HOSPITAL, MOOROOPNA, VICTORIA: Junior Resident Medical Officer.

MOTHERS' AND BABIES' HEALTH ASSOCIATION, ADELAIDE, SOUTH AUSTRALIA: Medical Officer.

ROYAL AUSTRALIAN AIR FORCE: Medical Officer.

ROYAL HOSPITAL FOR WOMEN, PADDINGTON, NEW SOUTH WALES: Medical Superintendent.

THE BUNDARRA DISTRICT HOSPITAL, BUNDARRA, NEW SOUTH WALES: Medical Officer.

THE QUEEN'S (MATERNITY) HOME, ADELAIDE, SOUTH AUSTRALIA: Resident House Surgeon.

THE TOOWOOMBA HOSPITALS BOARD, TOOWOOMBA, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY Hospital, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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